TO STUDY PATTERN OF DERMATOLOGICAL MANIFESTATIONS OF OBESITY

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I hereby declare that the thesis entitled "**TO STUDY PATTERN OF DERMATOLOGICAL MANIFESTATIONS OF OBESITY**" submitted towards partial fulfillment of the requirements for the award of **MD in Dermatology, Venereology and Leprosy** degree to BFUHS is a compilation of original work carried out by me under the supervision of **Dr. Sunil Gupta**, Professor & Head, and **Dr. Sukhjot Kaur**, Associate Professor, Department of Dermatology, Venereology and Leprosy and **Dr. Naveen Mittal**, Professor, Department of Endocrinology. No part of this thesis has formed the basis for the award of any degree previously.

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Dedicated

to

my Family

&

Teachers

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Dr. Amandeep Singh

LIST OF ABBREVIATIONS

AN	-	Acanthosis nigricans
AN	-	Acanthosis nigricans
Acc.	-	According
BMI	-	Body mass index
CO2	-	Carbon dioxide
DVT	-	Deep vein thrombosis
FFA	-	Free fatty acids
FBS	-	Fasting blood glucose
HDL-C	-	High-density lipoprotein cholesterol
HIV	-	Human immunodeficiency virus
HS	-	Hidradenitis suppurativa
HPE	-	Histopathological examination
IR	-	Insulin resistance
IV	-	Intravenous
IgG	-	Immunoglobulin G
LDL	-	Low density lipoprotein
MRSA	-	Methicillin resistant staphylococcus aureus
MRI	-	Magnetic resonance imaging
NHANES	-	National health and nutrition examination survey
ND-YAG	-	Neodymium doped yttrium aluminium garnet
NIDDK	-	The National Institute of Diabetes, Digestive and Kidney Diseases
OCP	-	Oral contraceptive pill
PDL	-	Pulsed dye laser
RBC	-	Red blood cell
SC	-	Stratum corneum

SD	-	Standard deviation
TG	-	Triglyceride
TNF-α	-	Tumor necrosis factor-alpha
TEWL	-	Trans-epidermal water loss
TC	-	Total cholesterol
US	-	United states of America
VVC	-	Vulvo-vaginal candidiasis
WHO	-	World Health Organization
WHR	-	Waist-hip ratio
WC	-	Waist circumference
XP	-	Xanthelasma palperum

ABSTRACT

Background: Obesity has reached epidemic proportions in many parts of world. Obesity is associated with an increased risk of development of various health problems like hypertension, diabetes mellitus type II, dyslipidemias along with various skin manifestations which can be diagnosed early.

Objective: To study the pattern of various dermatological manifestations in patients with obesity

Methods: This was an observational study conducted at Department of Dermatology and Endocrinology, Dayanand Medical College and Hospital, Ludhiana. A total of 250 patients with BMI>25kg/m^2 were included. Anthropometric measurement with fasting lipid profile and FBS were done. Patients were divided into obesity grade I & grade II and cutaneous examination was done.

Results: Out of 250 patients 168 were of grade I and 82 of grade II obesity. There were many patients with multiple manifestations, so in total there were 354 dermatoses. The most common finding was acrochordons (24.58%) followed by Xanthelasma (11.30%), follicultitis (9.89%) and other manifestations with slight female preponderance (51.60%).

Conclusions: Obesity is commonly associated with wide range of dermatological manifestations. Identification and treatment at an early stage can prevent long term cutaneous complications.

Key words: Obesity, dermatological manifestations, lipid profile, fasting blood glucose

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INTRODUCTION

Obesity is a state of excessive amount of body fat associated with an increased risk of medical illness and pre mature death.[1]The World Health Organization (WHO) classifies “overweight” as a body mass index (BMI) of 23-24.9 kg/m^2 and obesity as a BMI of ≥25 kg/m^2 for asian population.[2] Morbidity and mortality in obese population is significantly more than the normal weight individuals. Regional fat distribution may have an effect on risks associated with obesity. According to The National Institute of Diabetes, Digestive and Kidney Diseases (NIDDK) , women with waist–hip ratios of more than 0.8, and men with more than 1.0 , are at increased health risk because of their fat distribution. Cut off for waist circumference for South Asian men and women are more than 90 cm and more than 80 cm respectively. [3]

In the recent years, obesity has received a considerable attention as a major health risk, to the point that some scientists have even predicted that it could become responsible for reversing the current trend of increasing life expectancy.[4] Obesity is the most prevalent form of malnutrition in the industrialized countries, and it is rapidly becoming highly prevalent in the developing world. It is now considered as a major contributor to the global burden of disease and disability.[5]

This increase in the prevalence of obesity in general population is accompanied by increase in many other co-morbidities which are associated with obesity and are less frequently seen in population with normal BMI.[6]

Obesity is now emerging as an important health problem in many developed and developing countries. Approximately 30% of Americans are obese.[7]Worldwide, the prevalence of obesity varies from 7% in France to 32.8% in Brazil.[8] Obesity is most common in women and the elderly, but is increasing in children. A Chinese study reported a prevalence of obesity in children ranging from 4.3% to 17.4%.[9] This recent increase in obesity is due to increased calorie intake and a decrease in physical activity among population.The prevalence of overweight and obesity in India is increasing faster than the world

average. For instance, there has been an increase in the prevalence of overweight from 8.4% to 15.5% among women between 1998 and 2015, and the prevalence of obesity increased from 2.2% to 5.1% over the same period.[10-12]

It can result from increased energy intake or decrease in expenditure or a combination of both. Personal habits, eating habits, genetic factors, and various neuropeptides play an important role in development of obesity. Prevalence of obesity in a particular population can be considered as a rough indicator of its health status.[13] Obesity is becoming a problem in countries where various changes in socioeconomic status, lifestyle changes and habits have led to increased food consumption and a decline in physical activity. [14]

Obesity is commonly accompanied by various other co-morbid conditions such as hypertension, diabetes mellitus type II, cardiovascular disease, and is itself a major risk factor for respiratory disorders, endocrine disorders & psychiatric disorders.[6] It is indirectly related to anxiety, impaired social interaction, and depression.[14]Obesity is also a part of metabolic syndrome, which is characterised by insulin resistance and an increased risk of cardiovascular diseases and is associated with other systemic disorders.

Various dermatological changes have been reported in patients with obesity. It can be attributed to increased weight, insulin resistance, over distension, excessive fat folds leading to increased humidity and maceration over folds.

Obesity causes abnormalities in several functions of the skin, like skin barrier function and sweat production, collagen structure and function, wound healing, microcirculation and macrocirculation.Dermatological changes have been classified according to pathophysiologic origin as being associated with insulin resistance (IR), hyperandrogenism, skin folds, mechanical causes, and hospitalization.[15,16]

A number of significant skin complications of obesity such as:

- Plantar keratosis

- Acanthosis nigricans
- Striae
- Skin tags
- Keratosis pilaris, could be considered as cutaneous stigma of severe obesity.[15]

There is characteristic peripheral insulin resistance that leads to hyperisulinemia, which is considered as a root cause of obesity. Hyperinsulinemia is implicated in causation of type 2 diabetes, hypertension, dyslipidemia, coronary artery disease, and some skin diseases, such as acne, acanthosis nigricans.[14]

Obesity is also associated with increased death rates.[17] The causes of obesity are diverse and often involve combinations of several etiologies. Primary endocrine disease is rarely the cause of obesity; however, hyperinsulinemia and diabetes are well-known consequences of obesity. The genetic predisposition to obesity is considered to involve multiple genes, these genes in a “permissive” environment predispose an individual to excess adiposity. There are many single-gene defects that present with severe early onset obesity; like leptin deficiency and abnormalities of the melanocortin receptor.[18]

Overweight and obesity depend on the imbalance between energy intake and expenditure, and food intake is in turn controlled by internal signals.[19]

Obese children are at higher risk of precocious puberty, polycystic ovary syndrome, sleep apnoea, orthopedic complications, and psychological and social disturbances.[20] These children have a higher probability of becoming obese adults.[21]

Obesity is associated with multiple metabolic alterations and it is believed that insulin resistance in the adipose tissue, liver, and skeletal muscle is crucial in the pathogenesis of these metabolic abnormalities.[22-24] Adipocytes act as key regulators of whole-body energy homeostasis, and altered adipose tissue glucose metabolism is an important cause of insulin resistance and metabolic function.[25] Adipose tissue contributes to the development of glucose abnormalities

through excessive release of free fatty acids (FFA), adipokines, cytokines, and macrophage infiltration.[26]

Obesity has a significant effect on lipoprotein metabolism. Typical plasma lipid abnormalities associated with visceral obesity are increased plasma concentration of triglycerides (TG), reduced concentration of high-density lipoprotein cholesterol (HDL-C), and quantitative alteration of low-density lipoprotein (LDL) fraction.[27]Obese patients, in the presence of abdominal obesity or visceral fat accumulation, are frequently characterised by a particular dyslipidaemia with high triglycerides and low HDL cholesterol levels. There is a clustering of prediabetes/diabetes, hypertension, hypertriglyceridaemia and low HDL cholesterol levels in patients with abdominal obesity. This cluster is labelled as metabolic syndrome.

Various skin manifestations are common to a large proportion of obese patients, such as striae distensae, plantar hyperkeratosis and an increased risk of skin infections.[28] Cutaneous infections, including methicillin-resistant Staphylococcus aureus, are more common in those with an elevated BMI.[29] This may relate to the physical effects of larger skin folds and equally relate to the immunological alterations seen in the skin in the obese individuals. Poor mobility and inability to maintain adequate levels of hygiene may exacerbate this problem.

Various systemic diseases have been linked to secondary dyslipidemias and xanthoma formation which include nephrotic syndrome, hepatic abnormalities, and thyroid dysfunction. Xanthomas can be due to:

- Primary dyslipidemias, may carry a heritable or familial component
- Secondary dyslipidemias, which are typically acquired but mayunmask an underlying genetic predisposition
- Normolipemic states in which there is no underlying dyslipidemia and is relatively rare compared to others.

The vascular supply is impaired in obese persons. [30]Obesity is associated with elevated blood pressure and systemic vascular

resistance responses to mental stress. Normo-tensive obese individuals shows a markedly impaired skin and muscle microcirculatory responses to mental stress. This increased propensity of obese individuals to develop hypertension under conditions of chronic psychosocial stress may be responsible for obesity-related hypertension and cardiovascular disease. In non-diabetic obese patients, there is a significant reduction in the cutaneous vasoconstrictive response to sympathetic activation.[31]

Obesity impedes lymphatic flow, which leads to collection of protein-rich lymphatic fluid in the subcutaneous tissue. This accumulation frequently results in lymphedema which is associated with dilatation of tissue channels and reduced tissue oxygenation.[16] Accumulation of fluid in the setting of decreased oxygen tension leads to fibrosis and a chronic inflammatory state.

Intertrigo is a manifestation of obesity that involves a combination of skin irritation and infection involving the body folds. Predisposing factors include friction, maceration, moisture, warmth, sweating, and occlusion.[32] Skin of patients with BMI greater than 25 is more alkaline than that of patients with normal BMI, predisposing these overweight and obese patients to superinfection by Candidal species. [33]

Colonization with bacteria, yeast, and dermatophytes may be responsible for exacerbation. Areas which are generally involved may include genitocrural, subaxillary, area below the abdominal fold, gluteal, and submammary skin folds.

Cellulitis is common over legs with coexisting lymphedema. This cellulitis can be difficult to clear, as it may require higher doses of antibiotics than used normally because of the higher BMI and also need prophylactic antibiotics to prevent recurrence.[34]

Folliculitis, furunculosis and paronychia are other problems encountered by many and increased incidence of Candida albicans infection is also associated with obesity.[28]

Plantar hyperkeratosis due to increased weight.[15] The shape of the foot changes in obese individuals. Obese children have a lower footprint angle and obese individuals have a wider forefoot width and

higher plantar pressures during standing and walking.[28]There is an abnormal transference of weight during walking that alters the alignment of the foot, causing increased stress over bony prominences. Plantar hyperkeratosis is a part of the skin's protective response to increased mechanical stress. It can create many problems including impingement of the plantar nerves, arthritis of the metatarsophalangeal joints, painful ambulation, impaired balance, and difficulty wearing shoes. [35]

Striae distensae, characterized by linear, smooth bands of atrophic-appearing skin that are at first reddish, then purple and finally white, when they become depressed; are closely associated with obesity.[36]They generally follow cleavage lines perpendicular to the direction of greatest tension and occur most commonly on the abdomen, buttocks and thighs, as well as the inguinal region. Striae can be regarded as scars that result from dermal connective tissue injury in which the newly generated collagen aligns in response to local stress forces.[37]

Acrochordons are frequently seen in association with acanthosis nigricans, these present as pedunculated soft brown papules most commonly seen on the neck and in the axillae and groin[14] Classically found in the flexural areas such as the nape of the neck, the axilla, and the groin; acrochordons can be observed on many other surfaces such as the vulva and the areola. These are commonly present in Native Americans, African Americans, and Hispanics.[38]

Acanthosis nigricans appears as symmetric, velvety, hyperpigmented plaques commonly observed in the axilla, groin, and posterior neck but can also be seen on the elbows, knuckles, and face.[14]Biochemical mechanisms for developing acanthosis nigricans involve significant insulin resistance at the cellular level along with compensatory hyperinsulinemia.[14]

Keratosis pilaris is a skin condition characterized by palpable sterile papules and/or pustules which affects approximately 16% of the population.[6] Common sites of involvement are the posterior aspect of the upper arms, anterior thighs, face, and buttocks; however, the

lesions of keratosis pilaris can occur anywhere except for the soles and palms. While keratosis pilaris is a benign condition, these lesions are associated with significant cosmetic comorbidity with 69% of patients reporting distress as a result of their rash.[6]

Obesity diminishes perfusion to the injured tissue leading to impaired wound healing.Leptin, the adipocytokine, promotes wound healing as well as inhibiting feeding and other immunological roles.[1, 39] Leptin resistance, associated with obesity, may contribute to the pathophysiology of impaired wound repair.

Being aware of such clinical signs and the underlying systemic disorders may facilitate earlier diagnoses, thereby permitting early therapy initiation and prevention of long-term sequelae of obesity. Understanding of these disorders is necessary both for the physicians as well as for the dermatologists, as many of these conditions are treatable, leading to an improved quality of life and reduction in psychological distress.

AIMS AND OBJECTIVES

1. To study the pattern of various dermatological manifestations in patients with obesity.

REVIEW OF LITERATURE

Obesity is generally defined as a condition of abnormal/excessive fat accumulation that may impair health.[1] The words 'obese'/'obesity' is derived from French and Latin, where 'obedere' means 'over eat' and 'obesitas' means being very fat.[40]

Obesity is a development-driven malnutrition that is rising among all ages and socio-economic groups. The prevalence of obesity seems to be increasing in most parts of the world, even where it used to be rare.[41] Environmental factors act as major contributor to the obesity epidemic, together with biological predisposition. Obesity develops in a situation where there is a positive imbalance between energy intake and energy expenditure, but the relative contribution of individual factors is poorly understood.

1. Dietary data from four consecutive NHANES studies, consisting of 39,094 adults in the USA, have shown that the temporal trends in the increase of the quantity and energy density of foods consumed by adults parallel the increasing prevalence of obesity in the US population.[42]
2. Data from the Central Statistical Office show that car ownership and television viewing, proxy measures of physical inactivity, closely parallel the rising trends in obesity in England.[43]
3. Using data from NHANES, Dietz et al. demonstrated that the prevalence of obesity increased by 2 % for each additional hour of television viewed.[44]
4. There is also evidence that the relative availability and price of different food products affect food consumption[45] and that the built environment, such as quality of local parks, affects the level of physical activities in a community.[46]

Obesity is now considered as a major contributor to the global burden of disease and disability, because of its association to various disabling conditions, such as heart disease, diabetes, hypertension, stroke, certain cancers, osteoarthritis, and respiratory abnormalities, and to several digestive diseases, including gastroesophageal reflux disease

and its complications, colorectal polyps and cancer, and liver disease [47] On a population level, a strong positive correlation between BMI and overall body fat content has been reported.[48]

Obesity and adipose tissue:

Various molecules which are secreted by adipose tissue play an important role in metabolic homeostasis, and their deficiency, caused by either massive adiposity or adipocyte dysfunction can lead to obesity or obesity-related pathologies. Adipose tissue is considered as an endocrine organ whose action on the target tissues depend on the location of fat depots and may differ according to the functional state. Leptin is one of the most potent adipokines involved in metabolic regulation by controlling body weight by signalling a sense of appetite to hypothalamus, which further produces neurotransmitters that lead to adaptation of food intake and energy expenditure. It also regulates hepatic lipogenesis by suppressing fatty acid synthesis[49] and improves muscle fatty acid oxidation. [50] Adiponectin, which is the most abundantly secreted adipokine plays a key role in enhancement of insulin sensitivity and partially reverse insulin resistance in obese mice. [51] Tumour necrosis factor-alpha (TNF-α) is involved in obesity-induced insulin resistance, it interferes with insulin signalling and blocks insulin actions. [52]

Obesity and Skin

Obesity can have a large number of detrimental effects on the skin. Skin of an obese patient has several physiological alterations which predisposes it to numerous pathologies. Various dermatologic changes have been reported in obese individuals, particularly in relation to the excessive fat folds that favours humidity and maceration along with bacterial and fungal overgrowth, striae which can be due to over-extension of skin, stasis pigmentation, which is usually related to peripheral vascular disease, acanthosis nigricans, and skin tags, associated with insulin resistance. [15]

Physiological Cutaneous Changes in Obesity

Skin barrier

A number of changes in barrier integrity have been linked to obesity these include xerosis and altered trans-epidermal water loss (TEWL). [53-59] Stratum corneum (SC) hydration changes in morbidly obese individuals is commonly associated with xerosis. In a cross-sectional study of 1300 patients, examination of SC moisture by evaluating skin capacitance showed obesity to significantly affect SC moisture. [54] Loffler et al found a higher TEWL in obese subjects; [55] which can be linked to profuse sweating which is commonly seen in this population. Increased TEWL was also found in another study involving obese children. [57]In contrast to above findings, one study showed TEWL to be lower in obese patients, [56] suggestive of a lower permeability of the skin to evaporative water loss which was attributed to the role of adipokines and leptin. Adipokines induces the replacement of the SC and enhance keratinocyte activation. [55,59] Leptin, a hormone which is predominantly made by adipocytes inhibits hunger, act as a mitogen factor for keratinocytes and also promote in vitro proliferation of fibroblast and collagen synthesis through signal transducer and activator of transcription 3 phosphorylation. [55,60,61]

Collagen structure and wound healing

In various animal models, obesity has been found to be associated with an altered collagen structure and impaired wound healing. [62,63] In obesity, deposition of collagen fails to go with the expansion of the skin surface area, which leads to decreased mechanical strength of the skin. [62] Leptin is responsible for wound healing, and obesity, which is associated with leptin resistance contributes to the pathological cycle of impaired wound repair. [36,39,64-65]

Sebaceous glands and sebum production

Sebum production plays a vital role in development of acne. [66-70] Androgens, insulin, growth hormone, and insulin-like growth factors are frequently elevated in obese patients and increases sebum production, thus affecting severity of acne. [16, 71]

Sweat glands

Obese individuals sweat more profusely as they have larger skin folds and thicker layers of subcutaneous fat. [55,56,72,73] Cramer and Jay in 2015 found that the higher rectal temperatures and evaporative heat balance is due to heat production, evaporative heat balance, and increased body size. [74] In addition, increased subcutaneous fat further increases the frictional and moisture components of the skin.[58] This humid environment leads to exacerbation of local inflammation, predisposing obese patients to various dermatoses, such as intertrigo, with overgrowth of bacteria, candidal species, and dermatophytes. [55,75,76] Obesity, diabetes, and intertrigo promotes bacterial overgrowth and the further development of bromhidrosis. [77]

Circulatory and lymphatic changes

Obesity leads to microvascular dysfunction, contributing to the development of microangiopathy and hypertension, [56,78] and this condition might worsen the clinical severity of chronic venous disease. [79] Obesity is related to venous insufficiency due to valve failure and high intra-abdominal pressure. [80-84]Venous insufficiency can cause varicose veins, varicose eczema and ulceration. [80-84] Cutaneous capillaries of obese patients are maximally recruited at rest, with no functional capillary reserve, contributing to the development of ulceration. [85] Furthermore, lymphatic flow is affected by increasing weight, which leads to lymphedema, dilatation of tissue channels, and reduced tissue oxygenation. Lymphedema further increases the risk of infection due to the collection of a protein-rich fluid in tissues. [36,86-88]

These physiological changes seen in skin of obese patients is responsible for various skin manifestations, we can now look into the various skin diseases which are related to obesity.

Striae Distensae

Striae distensae, is a common benign dermatologic ailment seen in obese patients that has the propensity to cause significant psychological stress for the patient. The psychological stress in association with striae distensae is frequently exacerbated by a sub-optimal response to standard treatment modalities. Common sites for

striae distensae appear to be abdomen, thighs, and lumbo-sacral region in both males & females, with breasts and buttocks more commonly involved in females. [89]

The clinical presentation can be divided into an acute and a chronic state with both of these frequently occurring simultaneously in the same patient. The acute state commonly known as striae rubra is characterized by flat, reddish finely wrinkled lesions which run perpendicular to tension lines of the skin. [90] If untreated these striae rubra lesions progress to a chronic state which is known as striae alba, presenting clinically as whitish, atrophic appearing, depressed, irregularly linear scar-like bands. [90]

Histology

Histologically, both striae rubra and striae alba have a characteristic appearance. Biopsy from striae rubra usually demonstrate epidermal edema with increased melanogenesis, vascular ectasia of papillary dermis, and reduced elastin with altered collagen in the reticular dermis with a characteristic reorganization of elastic fibers, with thicker fibers forming a peripheral border around a center of thin elastic fibers. [92]

The histological findings of striae alba shows epidermis which is characterized by the absent hair follicles, absence of melanocytes, decreased number of rete ridges, and generalized atrophy. The papillary dermis has decreased vascularity and the reticular dermis has deposition of thick bands of collagen parallel to the skin. Unlike histologic appearance of striae rubra which demonstrates a pattern of both thick and thin elastic fibers, histology of the reticular dermis of striae alba reveal lesions comprising primarily of thick fibers. [91]

Pathophysiology:

Striae in an obese patient is most often attributed to the mechanical changes which are associated with weight gain; however, striae can occasionally serve as the first sign of a more serious, systemic condition. Striae can occur due to hormonal change like in case of pregnancy, adolescence, and Cushing's Disease, in patients with an underlying dermal integrity disorder as in Ehlers Danlos, Marfan's, and zinc deficiency and patients using topical corticosteroids, especially

when applied under occlusive dressing. [92] The development of striae during pregnancy, known as striae gravidarum, is not related to the mechanical stretching of skin associated with pregnancy, but is primarily the result of the hormonal changes in association with pregnancy. [92] In patients with development of very rapid, multifocal striae in the absence of another obvious cause a possibility of cushing's disease should be kept. Simkin and Arce found in a study, higher levels of urinary adreno-corticosteroids in obese patients with striae as compared to obese patients without striae. [93] The clinical presentation of striae, however, was found to be lighter, narrower, and less atrophic than in those with Cushing's syndrome by Angeli et al. [94] Development of striae within body folds can be due to application of topical steroid because body folds act like an occlusive and increase the absorption of steroids by many fold. [95]

Treatment

Treatment of striae distensae often requires the use of multiple treatment modalities including topicals, dermabrasion, non-ablative & ablative laser therapy, and collagen induction therapy. The first step is to determine whether the patient is suffering from striae rubra, striae alba, or both because modalities such as tretinoin, which is quite effective for management of striae rubra but usually do not have a good effect in the treatment of cases of striae alba.

Initial treatment modality for patients with striae rubra is usually tretinoin 0.1%, which shows a significant improvement in both length and width of striae rubra.[89] Other topical agents, that can be used for the treatment as well as for the prevention of striae rubra includes olive oil, trofolastin, cocoa butter, and silicon gel. These agents have no significant improvement in striae or their development.[96]

In patients who do not respond to tretinoin therapy or those having striae alba, dermabrasion can be done.[97] The combination of glycolic acid 20% and tretinoin 0.05% is also efficacious in treatment of striae.[91]

Various laser therapy types that have good efficacy in the treatment of striae distensae include 585 nm pulsed dye laser (PDL), non-ablative

1320 nm Nd:YAG, and short-pulsed 10600-nm $C0_2$ Laser.[98] PDL is highly efficacious in the treatment of striae rubra where it shows an increase in collagen content in underlying skin as well as decreases the dilatation of blood vessels that act as a major contributor to the reddish discoloration of the striae. [99] Nd:YAG have been reported to help with re-pigmentation of striae alba. In patients with darker skin laser therapy should be avoided as the efficacy in this subgroup of patients is lacking and hypopigmentation of the treated areas may occur.[100]

Microneedling is a relatively safer option in which very fine needles are used to produce controlled injury to the skin by cutting microscopic channels into the affected areas which stimulate the body to heal the injury by collagen formation and neovascularization. It reduces the atrophic depressed areas of striae distensae without any risk of depigmentation associated with laser and intense pulse light therapy.In a recent study nearly half of patients treated with microneedling reported marked to excellent improvement, with all patients reporting at least minimal improvement. [101]

Acrochordons (Skin Tags)

Acrochordons, also known as skin tags, are brown or flesh-colored soft pedunculated papules which occurs in 25% of the general population. [102]There is an increase in incidence of acrochordons in the obese with a linear correlation between increasing BMI and an increase in the presence and density of acrochordons. Most common site of involvement for acrochordons include face, eyelid, neck, axilla, and groin; however, acrochordons at other sites like on the extremities, vulva, and a many other locations have been reported. [103]

The primary concern for the patient is cosmetic although some patients complaint of lesions to become painful and irritated secondary to the patient's clothing repeatedly rubbing against them. Obese patients often have large, dense, groupings of the lesions as compared to non-obese patients.

A study conducted by Gomez et al showed association of skin tags with obesity and high levels of insulin. [15]A study, by Puneet and

Deepak Mathurshowed that obese patients with skin tags had impaired tolerance of glucose in nearly 46% which further proves the interaction of IGF and glucose levels in pathogenesis. [104]A study by Omar El Safoury et al. showed that there were higher levels of leptin in patients with skin tags. [105] They also showed an increased mast cell concentrations near the lesions. In a study which was done in Egypt, presence of skin tags was seen in 64.2% of obese patients as compared to 52.1% in non-obese patients. [106]

On Histolopathological examination in case of acrochordons, there is presence of numerous dilated capillaries, as well as the varied loose connective tissue in stalk. [107]The epidermis of the acrochordons shows a combination of benign appearing acanthosis, hyperkeratosis, and papillomatosis. [107] Acrochordons are usually benign and in general their presence is not an indication of any serious underlying conditions, they can occasionally be associated with serious underlying conditions. This is true especially when encountering with the sudden and rapid development of multiple acrochordons in an obese patient as in case of Gardner Syndrome, which is a potentially fatal, heritable condition and is a subtype of familial colorectal polyposis. These are also associated with Birt-Hogg-Dube syndrome which is an uncommon heritable disease characterized by the presence of a triad of skin lesions: (1) acrochordons; (2) fibrofolliculoma; and (3) trichodiscoma. These patients have a seven times greater risk of developing renal malignancies than the general population. [108] Other associated conditions include Rabson- Mendenhall Syndrome, a rare heritable disorder caused by severe insulin resistance and Gorlin-Goltz syndrome, a rare heritable disorder characterized by the early occurrence of multiple acrochordons-like soft pedunculated basal cell carcinomas. Dermoscopy of these acrochordons-like skin lesions associated with Gorlin-Glotz syndrome reveals isolated blue-gray globules, small blue-gray ovoid nests, and fine elongated telangiectasias. [109] The association of the increased incidence of acrochordons with obesity, is the result of insulin resistance rather than

the increase in patient weight itself as with increased association of acanthosis nigricans with obesity. [104]

Treatment:

Various modalities that can be used to treat acrochordons:

1. Cryotherapy
2. Electrodessication
3. Excision with scissors.

Cryotherapy and electrodessication are associated with decreased incidence of bleeding; however, a major drawback is that healing of the thermal lesion may take several days.

Excision of small acrochordons with scissors can be done quickly without local anesthetic unless the lesion is large enough that the discomfort caused by the excision will exceed the discomfort caused by injection of local anesthetic.

Acanthosis nigricans

Acanthosis nigricans is a condition characterized by hyperpigmented, roughened, plaque which is described as velvety or leathery in appearance. Patient with acanthosis nigricans may also complain that the affected skin is malodorous. This is one of the most common dermatological condition seen in the obese individuals, affecting roughly 74% of obese patients. [38] Common sites of involvement include flexural areas such as the nape of the neck, axilla, and groin; however, it can also be seen on many other sites such as the vulva and the areola. It is mostly seen in Native Americans, African-Americans, and Hispanics. [38] There is a recent increase in incidence among children as a result of the epidemic of childhood obesity. Recent studies show that nearly a quarter of elementary school aged children with more than 120% of their ideal body weight are afflicted. [110] Diagnosis can be made clinically; however, on HPE, acanthosis nigricans is hallmarked by variable acanthosis, papillomatosis, and hyperkeratosis.[38]

Acanthosis nigricans is almost always a result of hyperinsulinemia secondary to tissue insulin resistance. There is a direct correlation between the degree of insulin resistance and the severity of the

disease in these patients. [111] Excess of circulating insulin over stimulates insulin-like growth factor receptors in the skin resulting in significant epidermal proliferation & this hyperkeratosis causes characteristic hyperpigmentation and velvety appearance. [111] Hyperpigmentation in AN is not the result of increased melanogenesis and histologically these lesions do not have an increased presence of melanocytes or melanosomes.

Even in case of known hyperinsulinemia, a serious systemic disease may co-exist. There are various other causes of acanthosis nigricans which include malignancy like gastric, liver, colon and ovarian adenocarcinoma, use of nicotinic acid, Cushing's disease, and hypothyroidism. On rare occasions, familial cases of acanthosis nigricans have been noted. [112]

Treatment:

Treatment modalities available for AN:

1) Topicals
2) Oral medications
3) Laser therapy
4) Surgery

In the case of obesity-induced insulin resistance, weight loss results in significant improvement to complete resolution of the skin abnormalities. Medications for treatment of insulin resistance include metformin and thiazolidinediones. [113] Octreotide can be used in patients with insulin resistance as it lead to decrease in secretion of insulin. Patients suffering from acanthosis nigricans secondary to insulin resistance will report a worsening of the skin symptoms upon starting insulin therapy.This phenomenon is seen in patients who fail to rotate injection sites and begin to develop new lesions at these injection sites.[114] Similarly , patients with acanthosis nigricans as a result of underlying malignancy often have resolution of the skin lesions once resection of the tumor is done.

0.1% tretinoin is successful at lightening the hyperpigmented lesions. Combination of tretinoin with ammonium lactate 12% is associated with greater improvement than with tretinoin used alone.[113] Irritation from

tretinoin use is a common side effect, so patients can be started with application of 0.025% tretinoin cream every other day with slow elevation to daily use and the increasing of concentrations.

In patients with failed tretinoin therapy, topical calcipitriol therapy can be used, which act by activation of vitamin D3 receptors thus decreasing proliferation of keratinocytes, resulting in lighter lesions. [115]

In case of failure of topical therapy, systemic therapy is started. Oral isotretinoin can be successfully used to treat acanthosis nigricans. The clinical improvement usually reverses upon discontinuation of the therapy, so most patients will require a daily dose for the maintenance of improvement.[35] In cases with failure of medical management, dermabrasion, long pulsed laser therapy or surgical excisions can be used.

Plantar Hyperkeratosis

Plantar hyperkeratosis is thickening of the skin over the metatarso-phalangeal joints, which is usually secondary to increased pressure and mechanical stress placed on feet, in particular the metatarsal heads, due to the weight gain associated with obesity. Hyperkeratosis is part of the physiologic protective response of skin to increased mechanical stress but it can create a number of problems like impingement of the plantar nerves, arthritis of the metatarso-phalangeal joints, painful during walking, impairment of balance, and difficulty in wearing shoes. [35] These abnormalities not only disrupt the patient's activities of daily living but also put the patient at an increased risk for falls. Keratoderma climactericum is a common form of acquired palmoplantar keratoderma which is frequently associated with obesity. [116,117]

A study, by Al Mutairi in 437 obese patients showed presence of plantar hyperkeratosis nearly 45 % of patients. [118]Horseshoe type hyperkeratosis over the posterior sole is considered as a marker for obesity. [119]

On histopathological examination there is thickening of the stratum corneum.

Treatment:

The primary approach for management of plantar hyperkeratosis includes reduction of body weight as this helps to improve the underlying cause of the lesion. [120]While the patient is losing weight, there are several other effective treatment modalities for improvement of symptoms.

Surgical debridement of the lesion can be done using a scalpel blade to remove the excess keratin which can provide almost immediate relief.[120] Other treatment options for plantar hyperkeratosis include

1. Lactic acid creams
2. Foot bath
3. Salicylic acid

While use of pumice stones and other callus removers is successful in some patients**,** the risk of ulceration in a diabetic patient or the patient with peripheral neuropathy usually outweighs the potential benefit.

Intertrigo

Intertrigo is an inflammatory skin condition that occurs in body folds as a result of the friction between opposing skin, the localized moisture, and the occlusion due to these folds. There is usually an infectious component in intertrigo. These conditions together produce an area with intense erythema and maceration that often is so severe that these lesions weep and crust. Most common sites of involvement of intertrigo are groin, the inframammary area, and the axillae.[33,121] It can also involve inter-digital web spaces.

Obese patients can present with intertrigo between folds which are created by the obesity itself. Intertrigo has a strong association with obesity with a direct correlation between the incidence of the disorder and the weight of patient.

1. Obese individuals have increased surface area of body folds thus providing more skin area for friction to occur.
2. Obese patients have been found to sweat more in these body folds and they retain more moisture within the folds.
3. Skin of these obese patients with a BMI of more than 25 has been found to be more alkaline than that of patients with normal

BMI predisposing these overweight and obese patients to superinfection by various candida species. [63]

Poor mobility and the inability to maintain adequate levels of hygiene lead to exacerbation of this problem.[122] Presentation of intertrigo depends on the location of these lesions. Patients with lesions in body folds regularly complain of pain, burning, and itching; emotional distress as a result of the cosmetic morbidity caused by the lesion. Patients with intertrigo of the toe web spaces are either asymptomatic or complain of a burning sensation.

Evaluation of patients with intertrigo can be done in two steps:

First, distinguishing lesions of intertrigo from other similar appearing lesions; and secondly, ruling out any secondary infection of the lesion. Intertrigo can be diagnosed clinically based on the characteristic appearance of the lesions.Patients that are refractory to treatment should be carefully evaluated for possibility of other conditions that may mimic the clinical presentation of intertrigo.

C. albicans infections are more prevalent in patients with diabetes mellitus because of a higher pH in intertriginous areas. It acts as a major factor that promotes the susceptibility of patients with diabetes mellitus to Candidal infection of the skin. [33]

KOH preparations are very useful in confirming the presence of candida in lesions with the presence of satellite papules and pustules which are generally considered pathognomonic for candida superinfection.[123]Other secondary infections commonly seen in obese patients with intertrigo include:

Streptococcus pyogenes, Staphylococcus aureus, Proteus species, *Pseudomonas aeruginosa*, Dermatophytes, and *Corynebacterium minutissimum*. [123] Wood's lamp examination will reveal a characteristic green fluorescence with pseudomonas infection and a coral-red fluorescence with infection with *Corynebacterium minutissimum*.[28]Clinical signs indicating infection with Group A Streptococcus include a foul smell, lack of characteristic papular or pustular lesions, and a presence of delineated, fiery red lesion.[123]

Bacterial culture can be done for determination of the specific cause of secondary infection and antibiotic sensitivity test to help guide subsequent therapy.

Treatment:

In patients with a secondary infection with candidal species topical antifungals like azoles, terbinafine, etc. and Greer's Goo which is a combination of nystatin powder, hydrocortisone powder, and zinc oxide paste have been effective. [123]

In cases which are resistant to initial therapy with topical treatments, oral fluconazole can be used. Castellani's carbol fuchsin paint has been effective for patients suffering from intertrigo of the toe webs. [28] It is very important to aggressively treat secondary candidal infections to avoid any subsequent development of onchomycosis.

With bacterial infections, treatment can be started empirically; however, it is important to adjust accordingly based on antibiotic susceptibilities. A secondary infection with Group A Streptococcus infection can be treated with a combination of oral amoxicillin-clavulanate combined with topical application of fusidic acid cream.

Bacterial infections:

Obesity may be associated with a multiple bacterial infections, ranging from less complicated infections such as folliculitis and furunculosis[124] to more serious infections including erysipelas[125-126] and necrotizing fasciitis[127-130] that require hospitalization.

Erysipelas, most commonly caused by streptococcal species and is known to complicate lymphedematous limbs. [34] Obesity without lymphedema has also been proven as an independent risk factor for erysipelas.[126] Cellulitis can occur particularly in legs with coexisting lymphoedema. This cellulitis is difficult to treat, as it may require higher initial doses of antibiotics and may also need prophylactic antibiotics to prevent recurrence. [131]

Necrotizing infections of the skin includes necrotizing cellulitis and necrotizing fasciitis. Necrotizing fasciitis is a deep gangrenous infection of subcutaneous tissue that leads to progressive destruction of fat and

fascia, but occasionally spares the skin.[132]Necrotizing cellulitis and necrotizing fasciitis is generally characterised by an extensive tissue destruction, systemic toxicity, and high mortality. Other features include thrombosis of blood vessels, bacteria spreading along fascial planes, and limited infiltration of acute inflammatory cells. In a study done by Gallup et al, [127] 88% of women hospitalized for necrotizing fasciitis were obese and more than 3 risk factors, including obesity, diabetes, hypertension, and advanced age, were predictive of 50% mortality due to this rapidly progressive infection. [130]

Necrotizing fasciitis should be considered as a possibility in the setting of fever, toxicity, skin involvement with pain out of proportion to skin findings, and elevated levels of creatine phosphokinase. In case of suspicion of necrotizing fasciitis, surgical exploration with appropriate cultures should be done to make the diagnosis. Punch biopsy for frozen section can be done to establish the diagnosis. [132]

Treatment:

Treatment include aggressive surgical debridement of all necrotic tissue, nutritional support, and broad-spectrum antibiotics. [131,132]

Pyoderma

Pyoderma is defined as any bacterial infection of the skin. They can be primary pyodermas which includes impetigo, furuncle, folliculitis, carbuncle, abscesses, sycosis barbae, erysipelas and cellulitis, secondary pyodermas which follow an underlying infective or eczematoid disorder and include scabies, eczema, traumatic wound and intertrigo.

The ‘PROCEED’ conducted study by Wolf et alshowed that obese individuals had 8% increase in soft tissue infections as compared to non obese.[133]A study conducted in Kentucky, USAshowed that nearly 50% of patients admitted for skin infections were obese. [134]

Folliculitis

Folliculitis is defined as a bacterial infection that arises in a hair follicle.

Aetiology

Staphylococcus aureus is one of the commonest organism causing folliculitis, is a normal dweller of the nares in 20-40% of people.

Compromised epidermal barrier and decreased immunity due many coexistent conditions like obesity, diabetes, HIV, xerosis and eczema can predispose an individual to folliculitis. Occlusion and perspiration also act as aggravating factors.

Superficial folliculitis, also known as Bockhart's impetigo, starts as a small dome like pustule occurring at the infundibular ostia of a hair.

Periporitis Staphylogenes is a rare type of superficial folliculitis occuring due to secondary infection of a simple miliaria lesion in neonates caused by Stapylococcus aureus.

Hot Tub Folliculitis, a form of folliculitis seen in persons with practice of using hot bath tubs, caused by Pseudomonas aeruginosa. [135]

Deep folliculitis

Also known as sycosis barbae, sycosis vulgaris or barbers itch, is associated with perifollicular inflammation seen over beard area and upper lip and have a chronic relapsing course. Starts as erythema and itching over the upper lip region. In a few days pustules develop over the area which are pierced by hairs. Lesions rupture on shaving or manipulation leading to formation of an erythematous spot which further acts as origin for crop of lesions.

Chronic folliculitis of lower legs, is a chronic inflammatory disorder over lower legs which heal without scarring but are a major problem as they are persistent and resistant to conventional treatment.

Furuncle:

Furuncle is a common bacterial skin infection seen in all ages and in all regions of the world. It presents as an inflammatory nodule that is deep seated and developing from a preceding superficial folliculitis around a hair follicle. It is more prevalent in the pubertal group.

Aetiology

Staphylococcus aureus is the most common organism causing this infection. Altered balances in the normal skin flora and defective skin defence mechanism lead to clinical infections. Occlusion, friction, shaving and perspiration are predisposing factors for the infections. Various risk factors for extensive and disseminated lesions include, malnutrition, obesity, immunosuppressed states like HIV, diabetes,

usage of chemotherapeutic drugs, defects in neutrophil function and blood dyscrasias. Staphylococcal nasal carriage is an important cause for recurrent infections.

Clinical picture

It start as a small inflammatory nodule with erythema which progresses through stages of pustulation and necrosis to heal with extrusion of the necrotic content. Tenderness is usually associated with nasal or external ear canal lesions. In patients with malnutrition or other immune-suppressive states dissemination of septic foci can lead to septicaemia and pyaemia. Common sites of infection are face, arm, neck, finger, wrist, genitals and buttocks.

Treatment of pyodermas

Personal hygiene is one of the most important factor in preventing and controlling infections. Warm compresses may help to reduce inflammation. Deep lesions, need to be drained with incision and drainage, superficial lesions can drain spontaneously. Topical antibiotics like clindamycin, mupirocin, bacitracin and fusidic acid are effective for the condition. Oral antibiotics act as mainstay for treatment of bacterial infections. Cephalosporins, penicillinase resistant penicillin like cloxacillin, flucloxacillin are commonly used. Drugs like oral clindamycin, cotrimoxazole, rifampicin, minocycline and linezolid can be used in case of MRSA infections. IV linezolid or IV vancomycin can be used in cases of severe MRSA infections. [136]Topical application of mupirocin 2% to the anterior nares, oral rifampicin 600mg daily, ciprofloxacin 500mg daily or clindamycin 150mg for 3 months can prevent recurrences and persistent carriage.[137]

Erythrasma

It is a mild superficial bacterial infection localised to the skin caused by *Corynebacterium minutissimum*, a gram positive rod shaped organism commonly found in the normal flora of skin. High temperature and humidity is probably the most important risk factor for the disease. Disease is common in obese patients due to the effects of occlusion and friction. Diabetes is another important risk factor. It is more common in males but also commonly seen in obese women.

Clinical Presentation:

Characterized by erythematous well-defined irregular patches with minimal surface scaling initially which progresses with time and the colour changes from red to a diffuse brown colour. Common sites of involvement are toe clefts, axilla, groin. One of the most common site of affection is the fourth interdigital toe web space. [138]Lesions are usually asymptomatic. Toe cleft infections can cause fissuring, maceration. Obese individuals have an extensive, chronic and prolonged disease.

Diagnosis:

Wood's lamp examination gives coral- red colour fluorescence. This is due to the presence of coproporphyrin-3produced by the Corynebacterium species. [139]

Treatment

Topical fusidic acid and clindamycin can be used with good results. Oral therapy is the main stay of treatment with oral erythromycin, the most effective drug. In unresponsive cases, oral tetracyclines and photo dynamic therapy are being used.

Cellulite

Cellulite presents as dimpled skin over the thighs and buttocks of patient. It is primarily due to changes in epidermis and dermis rather than the adipose tissue. It results from fatty distension of the superficial fascial system, which connects the dermis and deep fascia. Points of attachment to the dermis are tethered while the surrounding areas, they bulge, producing the characteristic cobblestone appearance. Pronounced cases, likely involves decreased connective tissue growth in the face of progressive fat accumulation. [140]

Ultrasonographic examination of the upper thigh and buttock shows that there is herniation of subcutaneous fat into the reticular and papillary dermis. There is an increase in water-binding dermal glycosaminoglycans.[141,142] Cellulite is not exclusive to obesity, but usually accentuated by it.

Treatment:

Various modalities include:

1. Topical application of aminophylline which is well tolerated, pharmacological, non-invasive treatment for cellulite.[143] It is a phosphodiesterase inhibitor, enzyme responsible for breaking down cyclic adenosine monophosphate. It acts as a diuretic, possible mechanism of action for its anticellulite action is by fat lipolysis.[143-145]
2. According to Collins et al, most of the benefits are probably due to the adjuncts of exercise, dietary modification, and increased water intake that are mostly recommended. [146]
3. An anticellulite product containing retinol, caffeine, and ruscogenine; mechanized physical massage. [147]

Lymphedema

Lymphedema develops due to an abnormal accumulation of protein-rich lymphatic fluid that occurs when lymphatic return is impaired. Without the appropriate treatment of this condition, this stagnant lymphatic fluid causes tissue channel dilatation, reduces oxygenation of tissue and provides a culture medium for bacterial growth leading to recurrent infections.

The onset is usually gradual, with swelling of the limb. The edema is painless, and swelling disappears overnight. Clinically at the beginning, there is soft, pitting edema.

Lymphedema occurs predominately in the obese patients and presents a serious quality of life changes to this special patient population, with over 75% of morbidly obese patients suffering from lymphedema at some time during their lifetime.[148] Obstruction of lymphatic return is due to weight of patient's soft tissue, resulting in localized accumulation of lymph which further causes mechanical compressive effects in the affected region which further causes vascular compromise, exacerbation of the problem, and in turn makes these affected areas susceptible to bacterial infection. Bacterial infection triggers an inflammatory response within the lymphatic system which causes scarring of these lymphatic vessels causing further obstruction and lymphedema. This process generally starts in the distal lower extremities and progresses proximally. Progression of vascular

compromise and lymphangitis leads to a characteristic deformity of the affected extremity known as elephantiasis nostra verrucosa occurs**.**

There are four stages of lymphedema:

Stage 0: Subclinical lymphedema- There are no gross findings in this stage, microscopic examination of affected tissue demonstrates histologic changes of lymphangiectasia which is secondary to the initial impedance of lymph transport.

Stage 1: Reversible edema [149] Pitting edema is present, which can be resolved by elevation and compression of the affected extremity. On histological examination there is presence of lymphangiectasia, but also there are early fibrotic changes and dermal edema.

Stage 2: Irreversible edema- With progression of reversible edema, swelling in these patients is so severe that it is non-pitting and feels hard on palpation. Histopathological examination shows progressive fibrosclerosis and adipocyte hyperplasia.

Stage 3: Elephantiasis nostra verrucosa- Patients have significant dermatological changes characterized by papillomatosis, hyperkeratosis, and wart-like plaque formation over the affected extremity. These lesions can crust and weep lymphorrhea making the area susceptible to infectious cellulitis.

Usually there is associated pain and fatigue caused by the effort which is required to move grossly enlarged extremities that limit the patient's ability to ambulate.

Rarely primary forms of lymphedema which are secondary to genetic defects in the lymphangiogenesis pathway can occur, but secondary causes of lymphedema like obesity-induced lymphedema are much more common. [150]

Several other diseases mimic lymphedema which include venous insufficiency, edema secondary to congestive heart failure, edema secondary to kidney failure, edema secondary to other systemic organ failure, myxedema, edema secondary to radiation therapy and lymphadenectomy, post-traumatic edema, and deep venous thrombosis.

Another presentation is development of solitary, slow growing lesions called massive localized lymphedema. These lesions are also derived from the lymphatic drainage obstruction caused by obesity and presents with polyp-like masses and are defined by peau d'orange appearance and the presence of non-pitting edema.[149] Histopathological examination is done to rule out liposarcoma and can be distinguished by the absence of adipocytes and atypical cells which are commonly associated with liposarcoma. Histologically, these lesions are characterized by the presence of lymphangiectasia as well as both dermal and epidermal thickening. [150]

Treatment:

Prompt and aggressive treatment of diffuse lymphedema, as well as massive localized lymphedema, is mandatory as these are prone to development of infection and can potentially progress to malignancy. The propensity for these areas of lymphedema to undergo malignant transformation into a lymphangiosarcoma is known as Stewart-Treves Syndrome and is commonly seen in patients who have undergone radical mastectomy, but can also occur in patients with any form of chronic diffuse lymphedema. [151]

Treatment is generally directed towards reduction of the limb girth and weight, and further prevention of infection.[152]Symptomatic improvement can be done with limb elevation and compression with pneumatic compression devices and compression stockings, which serves as mainstays of treatment.

For patients suffering from massive localized edema, surgical excision can be done due to the risk of infection and malignant transformation in such lesions. Compression, lymph drainage, and weight reduction are the other methods of treatment in chronic lymphedema.

Chronic Venous Insufficiency

Chronic venous insufficiency and associated dermatological sequelae are commonly seen in obese patients and these changes can be attributed to:

1. Obesity itself results in an increased intra-abdominal pressure which causes increased resistance to venous return flow

ultimately leading to venous valvular insufficiency creating a cycle of worsening venous insufficiency.

2. Chronic inflammatory processes which are triggered by obesity causes on-going damage to the affected veins leading to development of both insufficiency and a propensity for thromboembolism.[153]

Various risk factors for development of varicose veins include parity, prolonged standing, inherited factors, and female sex. [154,155] Brand et al. found that obesity, sedentary lifestyle, and hypertension are defining factors for varicose veins in women, whereas in men a history of smoking is a more important issue. [156] Callam has hypothesized that obesity act as a promoter of varicose veins, instead of a true primary risk factor. [157]

The increased hydrostatic pressure within the veins due to venous insufficiency causes fluid, cells, and intracellular components to extravasate into the surrounding tissue. Initially this process is compensated by increased lymphatic drainage from the affected area, the ongoing extravasation of fluid, cells, and intracellular components eventually overcome the ability of the lymphatics to compensate resulting in areas of pitting edema, warmth, hyperpigmentation, and scaling of skin. The warmth and erythema are due to an inflammatory response against the breakdown products of extravasated red blood cells and intracellular components. If untreated, a few patients suffering from chronic venous insufficiency progresses to develop stasis dermatitis. Stasis dermatitis develops as a result of:

1. Inflammatory processes against RBC breakdown products
2. Due to a susceptibility of affected skin to physical and chemical assault as a result of venous insufficiency. [158]

Stasis dermatitis can present clinically in an acute, subacute, and chronic form. The acute form is characterized by a rapid onset of intense pruritus and profound redness mimicking cellulitis. [158] There may be presence of a weeping transudate and crusting. The subacute form is characterized by the slower development of a brown discoloration of the skin that results from extravasation of hemosiderin

from red blood cell breakdown products. Repeated episodes of acute and/or subacute stasis dermatitis can lead to development of chronic stasis dermatitis which is characterized by a thickening of the skin, intractable dark brown discoloration, and presence of cyanotic red plaque.

Another outcome of venous insufficiency in some of these obese patients is development of varicose veins, which develops as a result of incompetency of venous valves in the legs leading to reflux and dilation of the superficial veins. These superficial veins appear as torturous, dilated blood vessels that appear to arise from within the skin itself. These lower limb varicosities can cause discomfort of the legs which is aching in nature, with burning and itching. The main complication is venous thrombosis; however, unlike DVT, there is a little risk of clinically significant pulmonary embolism. [159]

Development of venous stasis ulcer is another complication resulting from venous insufficiency. Patients are likely to develop ulcerations once either the perforating or deep venous systems of the leg have been compromised. Most common site for development of venous ulceration is medial malleolus, although they can be found anywhere in the distal lower limbs when trauma and/or infection incite the lesions.[160] Recent theories on the development of venous ulcers suggests a systemic cause for chronic venous insufficiency, such a leukocyte abnormalities or impaired fibrinogen activity. [161-163]These ulcers have the tendency to enlarge, even in the absence of any obvious inciting or exacerbating factor. Patients with venous stasis ulcers usually complains about a constant aching pain, exacerbated by prolonged periods of standing and relieved by elevation of the affected lower extremity. Secondary infection of these stasis ulcers further complicates the treatment and impairs healing. Even with an optimal treatment it can take up to several months for venous stasis ulcers to heal and even with treatment, nearly 50% of ulcers will recur. [164]

Treatment:

In patients with distal lower extremity swelling in the absence of any serious dermatological issues, compression stockings or bandages

combined with elevation of the affected extremity provides adequate symptomatic relief. Weight reduction is important for slowing progression of the disease.

Patients suffering from acute stasis dermatitis with lesions, characterized by significant weeping are managed with cool water compresses two to four times per day to decrease transepidermal water loss. Topical steroids can be used to decrease pruritus and redness, but should not be applied directly into any cutaneous fissures or onto open areas as this can impair the healing process.

For subacute phase, topical steroids can be used. Because of increased chances of co-existent bacterial infection, appropriate antibiotics should be given. Other treatment modalities for patients suffering from lower limb varicosities include compression, lifestyle modifications, sclerotherapy, surgery, radiofrequency ablation, and laser therapy. [164]

Conservative therapy combining the use of compression and elevation is the mainstay of treatment, but it is inferior to sclerotherapy and other treatment modalities in terms of both symptom management and cosmetic improvement. Only pain is relieved by compression.[164] In case of failure of compression and sclerotherapy or in severe cases, the combination of avulsion and vein stripping has successfully treated varicosities and prevent further recurrence.

Venous stasis ulcerations are managed with surgical debridement of the ulcer in conjunction with occlusive gauze dressings. Concurrent identification and treatment of infection and treatment of nutritional deficiencies is to be done to achieve healing. Other adjunct therapies, like aspirin and the phosphodiesterase inhibitor pentoxifylline, also help to improve associated vascular insufficiency and to enhance healing.[165,166]

Adiposa Dolorosa

Adiposa dolorosa, aka Dercum's disease, is a rare multisystem disease which is commonly seen in obese, perimenopausal females. It presents with

1. Generalized obesity

2. Multiple, painful lipoma-like masses
3. Psychiatric illness including dementia, depression, confusion, and emotional lability
4. Chronic fatigue[167]

Patients complaints of severe pain which is out of proportion to what would be expected given the physical findings. Most of cases appear spontaneously but occurrence of the disease in family members has been reported.[168] Palpation of lipoma like masses in these patients tend to elicit pain. Common sites involved are abdomen, thighs, and upper arms. The skin over these masses is characterized by dilated superficial veins.

Diagnosis of this disease is difficult as there is lack of inflammatory, autoimmune, or general laboratory markers that can help in diagnosis. Biopsy has a limited role for diagnosis as finding from masses are indistinguishable from histologic findings of the common lipoma. MRI is done for diagnosis which reveals the following pathognomonic findings:

1. Multiple ill-defined, oval shaped masses that appear to blush on unenhanced MRI
2. No enhancement following the administration of gadolinium contrast
3. Decreased T1-weighted signal
4. Increased water-sensitive sequences[169]

Ultrasound evaluation shows superficial oblong shaped subcutaneous fatty noduleswhich are hyper-echoic and do not produce an increased color Doppler echo.

Treatment:

NSAIDs, oral prednisolone, and simple analgesics such as acetaminophen usually fail to provide pain relief. Weight loss, even when successful; fail to decrease the pain in most patients suffering from this condition. Use of oral mexilitine may provide symptomatic relief.[170] Intralesional corticosteroid injections can be given under ultrasound guidance as patients often struggle to successfully localize their pain. The most effective modality is surgical excision or

liposuction.[171]The injection of interferon alpha-2b may help in reduction of pain.[172]

Hyperandrogenism and hirsutism:

Hyperandrogenism in obese individuals is the result of increased production of endogenous androgens due to increased volumes of adipose tissue which synthesizes testosterone and hyperinsulinemia which further increases the production of ovarian androgens. [173]

Hirsutism is the development of excess terminal hair in androgen dependent areas in women. Cutaneous virilism includes features like hirsutism, acne vulgaris, hidradenitis suppurativa, and androgenic alopecia. There is an association between cutaneous virilism, acanthosis nigricans, keratosis pilaris, and insulin resistance. [173]In a study by Ruutiainen et al, there was a significant co-relation between hirsutism and BMI independently of age and testosterone level. [174]

Treatment:

Main focus should be on control of insulin levels; weight loss, oral contraceptives, and anti-androgenic therapies.[175]In obese women with polycystic ovary syndrome, thiazolidinediones may improve insulin resistance and hyperandrogenism.[176]

First-line therapy in the medical management of hirsutism consist of a combination of oral contraceptives and spironolactone. Oral contraceptives act by:

1. Increase in circulating levels of sex hormone binding globulin thus decreasing the levels of free testosterone
2. They decrease ovarian androgen synthesis.

The effects of OCP's are potentiated by the concomitant use of spironolactone which blocks androgen activity at receptor sites. In patients with renal failure, hyperkalemia, conditions in which spironolactone cannot be given, finasteride can be used in combination with oral contraceptives. Finasteride acts by inhibiting 5-alpha reductase thus decreasing the conversion of testosterone to the more potent dihydrotestosterone. Spironolactone and finasteride should always be used in conjunction with oral contraceptives in childbearing age group as these can feminize male fetuses if taken during

pregnancy.[176] Laser therapy can be next step in unresponsive patients with hirsutism.

Psoriasis:

There is a significantly higher prevalence of obesity among psoriasis patients than in the general population.[177]Inverse psoriasis appears to be commonly associated with obesity and sometimes is indistinguishable from intertrigo in obese patients. [177] Studies from Utah Psoriasis Initiative suggests that obesity may be a consequence of psoriasis rather than a risk factor for triggering disease. [177]An impact on physical activity either due to the cosmetic impact of psoriasis or to the locomotor effect of psoriatic arthropathy, predisposes an individual to obesity.Obesity is associated with morbidity of psoriasis. [178-181]In a study by Naldi et al, BMI >30 was found to be associated with an odds ratio of 1.9. [182]Various other studies have also found a significant association between obesity and increased morbidity of psoriasis. [183,184] Progressive weight loss can produce a significant improvement in the severity of psoriasis. [185-189] Bariatric surgery can produce rapid remission in psoriasis which is a direct evidence favouring that obesity may be causal in psoriasis. [185,188,189] A clear association between psoriasis and obesity was found in a meta analytical study done by A W Armstrong et al in data from nearly 130 studies done worldwide. [190]

Cutaneous complications of treatments for obesity:

Treatments for obesity are also associated with cutaneous complications. Some of the established pharmacological therapies for obesity have also demonstrated cutaneous side-effects. For example, orlistat therapy with the development of a lichenoid drug eruption. [191]

Jejunoileal bypass surgery, performed for weight loss, is associated with various cutaneous features, including a pustular erythematous macular eruption with a neutrophilic infiltrate similar to Sweet's syndrome, [192-194] a non-pruritic papular eruption with IgG and C3 deposition, [192] and erythema nodosum. [195]

MATERIALS AND METHODS

STUDY DESIGN

This is a descriptive study of new patients attending the outpatient clinic in the Department of Dermatology and Endocrinology of Dayanand Medical College and Hospital, Ludhiana. Study was done from October 2019 to march 2021. Ethical committee clearance was obtained and informed consent was obtained in the regional as well as in English language.

INCLUSION CRITERIA:

Patients aged >18years,belonging to either sexes attending outpatient and inpatient in Department of Dermatology and Endocrinology of Dayanand Medical College and Hospital , Ludhiana, were included in the study.

EXCLUSION CRITERIA:

- Patients not willing to participate
- Pregnant women and patients on systemic corticosteroids were excluded from the study.

METHODS:

A total of two hundred and fifty clinically diagnosed cases of obesity were included in this study. After informed consent, a detailed history, a comprehensive general and systemic examination was collected followed by a detailed skin examination (including hair, scalp and nails). The demographic details of the enrolled patients were documented. Height (meters) and weight (kg) were measured and BMI was calculated using the formula weight (kg) / height(m)^2. Patients having BMI >25kg/m² were included in the study. Cases were labelled as obesity grade I and obesity grade II as per classification of BMI by WHO for Asians.[2] Cutaneous lesions were diagnosed clinically.

A detailed physical examination including recording of vitals; anthropometry measurements-height (in meters), weight (in kgs), waist circumference in cms (at level of umblicus) and hip circumference in cms (at the level of widest part of buttocks)was done.

Relevant laboratory investigations like fasting lipid profile, fasting blood sugar and other relevant investigations were performed when required.

Number of cases

A total of two Hundred and fifty cases were included.

Statistical Analysis

Data was described in terms of range; mean ±standard deviation (± SD), frequencies (number of cases) and relative frequencies (percentages) as appropriate. All statistical calculations were done using SPSS (Statistical Package for the Social Science) SPSS 21 version statistical program for Microsoft Windows.

OBSERVATION AND RESULTS

This observational study included two hundred and fifty patients with BMI > 25 attending out- patient clinic and in-patient of department of Dermatology, Venereology and Leprosy and department of Endocrinology at Dayanand Medical College and Hospital. These patients were selected on basis of inclusion and exclusion criteria. FBS, fasting lipid profile and anthropometric measurements including: waist circumference and waist-hip ratio were done for all subjects. Patients were grouped into two groups according to BMI into grade I and grade II. Study period was from October 2019 to March 2021.

AGE DISTRIBUTION:

In table 1 and figure 1- Age group varies from 18 years to 81 years, with maximum number of patients in the age group of 41-50 years (27.60%) followed by 31-40years (26%) and minimum number of patients in the age group of >70 years (2.80%). Minimum age was 18 years and maximum age was 81 years.

TABLE 1: AGE DISTRIBUTION OF CASES

Age (Years)	No. of patients	Percentage
18-20	10	4.00%
21-30	25	10.00%
31-40	65	26.00%
41-50	69	27.60%
51-60	49	19.60%
61-70	25	10.00%
>70	7	2.80%

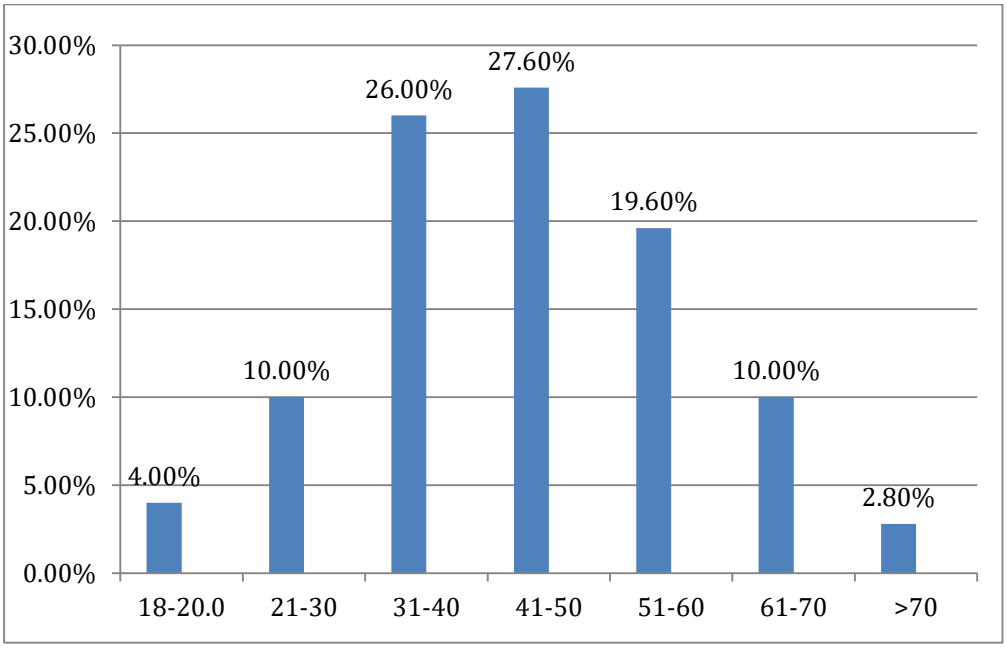

FIGURE 1: AGE DISTRIBUTION OF CASES

Gender distribution:

In table 2 and figure 2- out of total 250 patients, 129 (51.60%) were females and 121 (48.40%) were males.

TABLE 2: GENDER DISTRIBUTION OF PATIENTS

Gender	No. of patients	Percentage
F	129	51.60%
M	121	48.40%

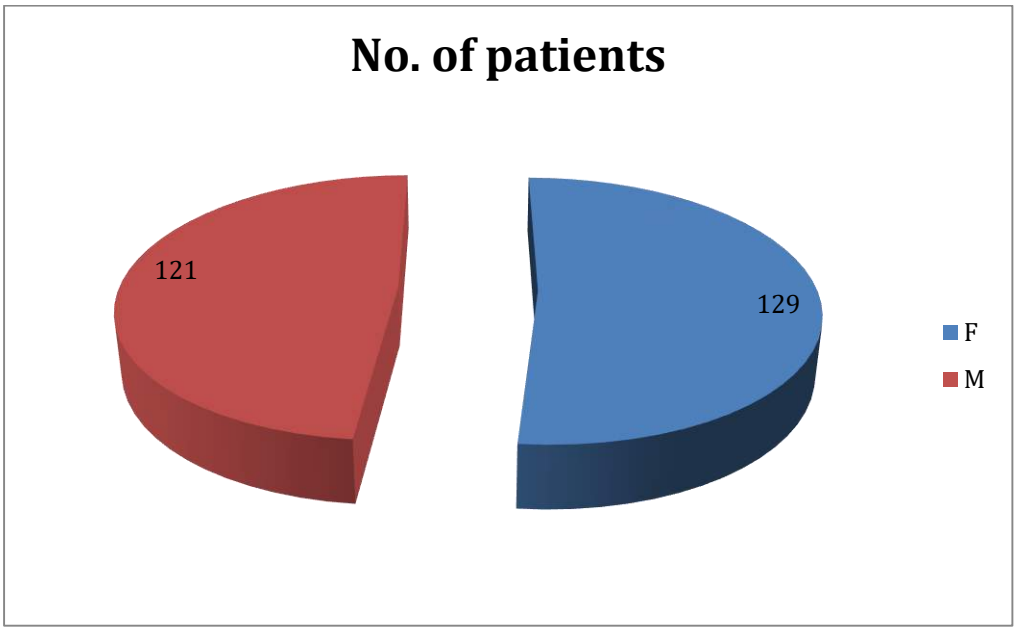

FIGURE 2: GENDER DISTRIBUTION OF PATIENTS

OBESITY GRADE OF PATIENTS:

In our study BMI was one of the important parameters used to select and classify patients. Patients were classified into obesity grade I and grade II as, obesity I (BMI25-29.9 kg/m^2), obesity II (BMI >30 kg/m^2). A total of 67.20% patients were having grade I obesity and 32.80% patients were having grade II obesity.

TABLE 3: GRADING OF OBESITY AS PER BMI

BMI GROUP	No. of patients	Percentage
I	168	67.20%
II	82	32.80%

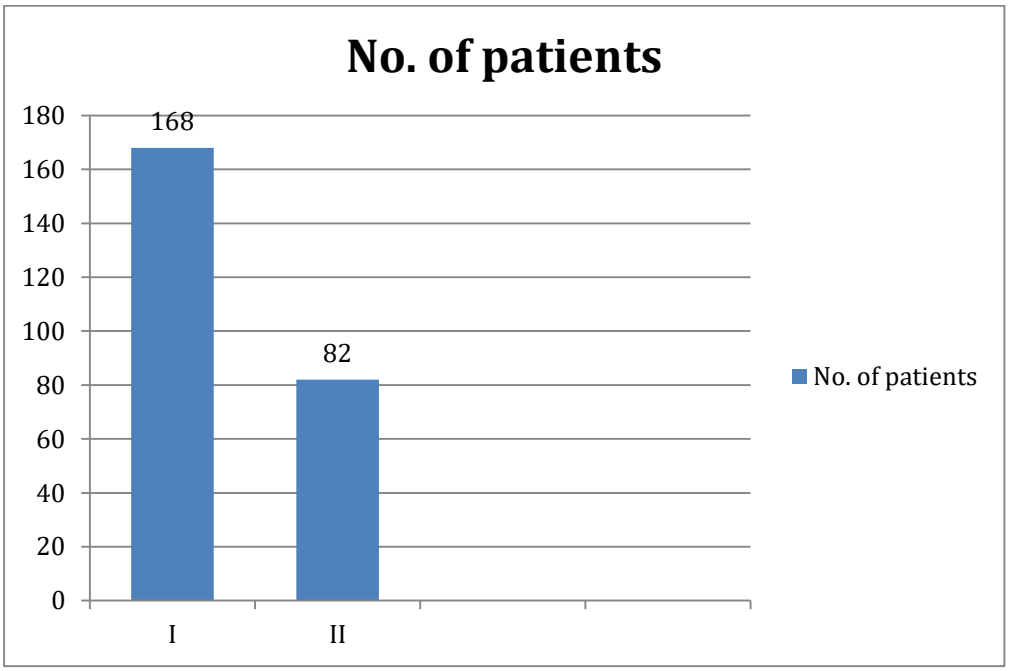

FIGURE 3: GRADES OF OBESITY ACCORDING TO BMI

WAIST CIRCUMFERENCE IN PATIENTS:

For study group, cut off for waist circumference for men and women was more than 90 cm and more than 80 cm respectively. Waist circumference was high in 90% patients and was normal in 10% patients.

TABLE 4: WAIST CIRCUMFERENCE OF PATIENTS IN STUDY

waist circumference(cm) GROUP	No. of patients	Percentage
Normal	25	10%
High	225	90%

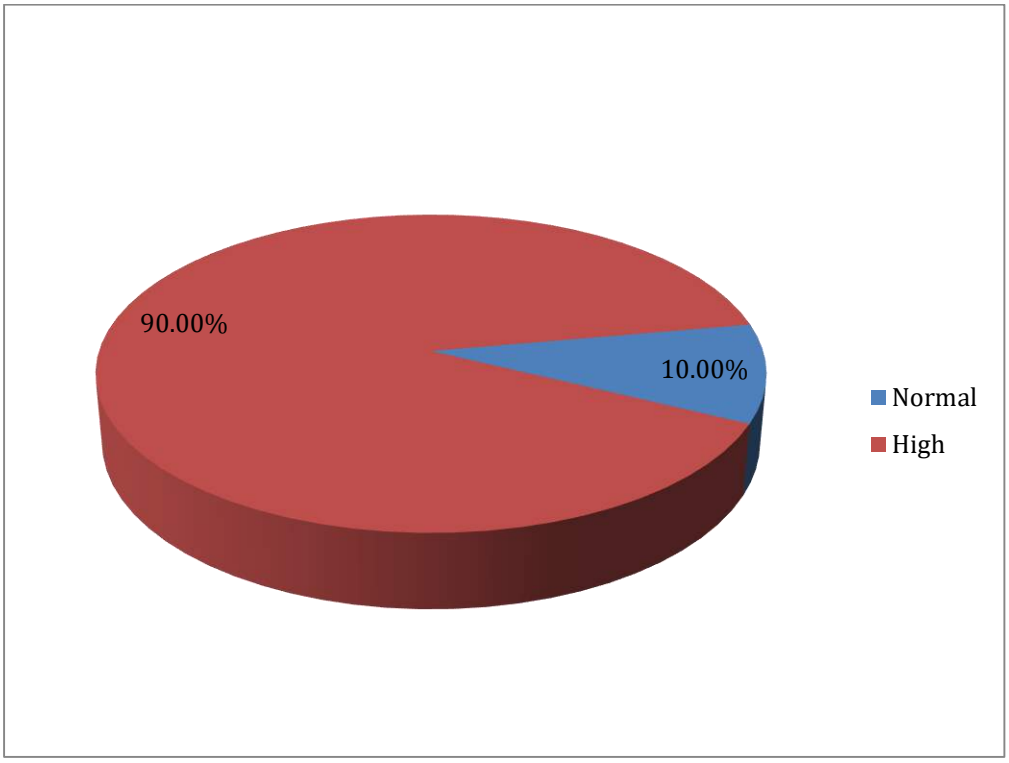

FIGURE 4: WAIST CIRCUMFERENCE IN STUDY GROUP

WAIST HIP RATIO IN CASES:

Cut off value for waist-hip ratio for men and women was taken as less than 1 and less than 0.8 respectively. Out of all 250 patients, a total of 71 patients (28.40%) had normal waist-hip ratio and 179 (71.60%) had high waist-hip ratio.

TABLE 5: WAIST-HIP RATIO IN PATIENTS IN STUDY

waist-hip ratio	No. of patients	Percentage
Normal	71	28.40%
High	179	71.60%

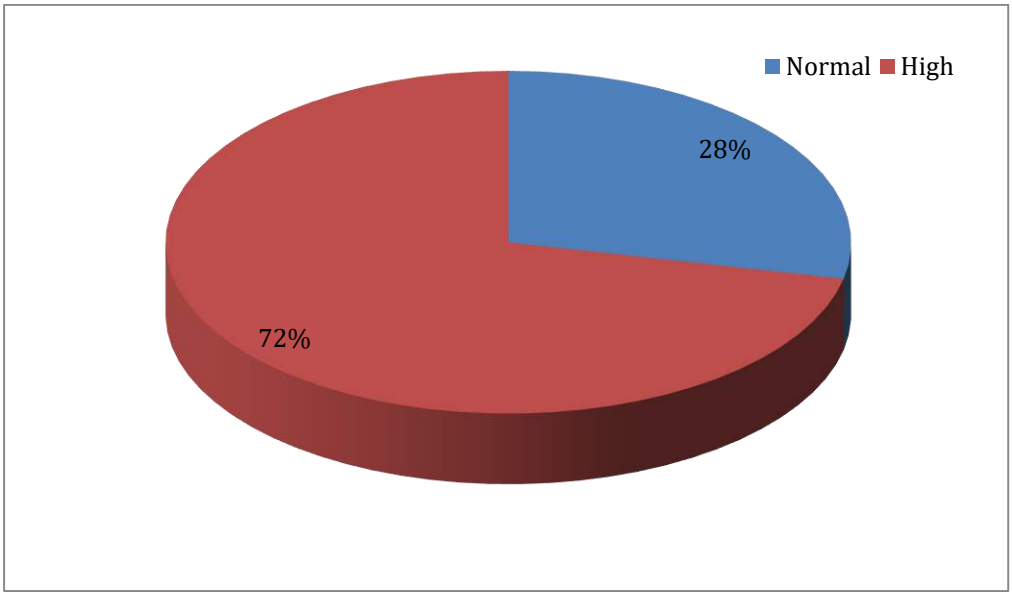

FIGURE 5: WAIST-HIP RATIO IN PATIENTS IN STUDY

LIPID PROFILE AND FASTING BLOOD GLUCOSE CHANGES IN PATIENTS:

Changes in lipid profile and fasting blood glucose associated with the dermatological disease were another major factors studied in our study. Out of 250 patients, total cholesterol was deranged in 74 (29.60%) patients; triglyceride and LDL were abnormal in 65(26%) and 64 (25.60%) patients respectively.

TABLE 6: LIPID PROFILE ABNORMALITIES IN PATIENTS

Total Cholesterol GROUP	No. of patients	Percentage
< 200	176	70.40%
> 200	74	29.60%
TG GROUP		
< 50	3	1.20%
50-200	182	72.80%
> 200	65	26.00%
LDL GROUP		
<80	60	24.00%
80-130	126	50.40%
> 130	64	25.60%

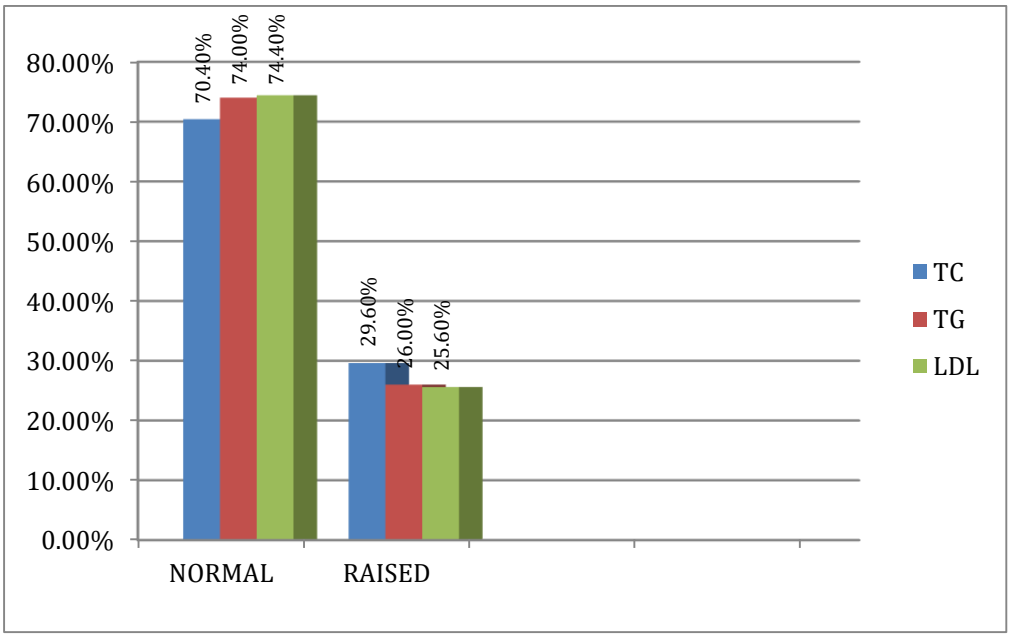

FIGURE 6: LIPID PROFILE ABNORMALITIES IN PATIENTS

Fasting blood glucose, which was also monitored in our study, was deranged (>110) in 53 patients (21.20%).

TABLE 7: FASTING BLOOD GLUCOSE IN PATIENTS

FBS(mg/dl)	No. of patients	Percentage
< 110	197	78.80%
> 110	53	21.20%

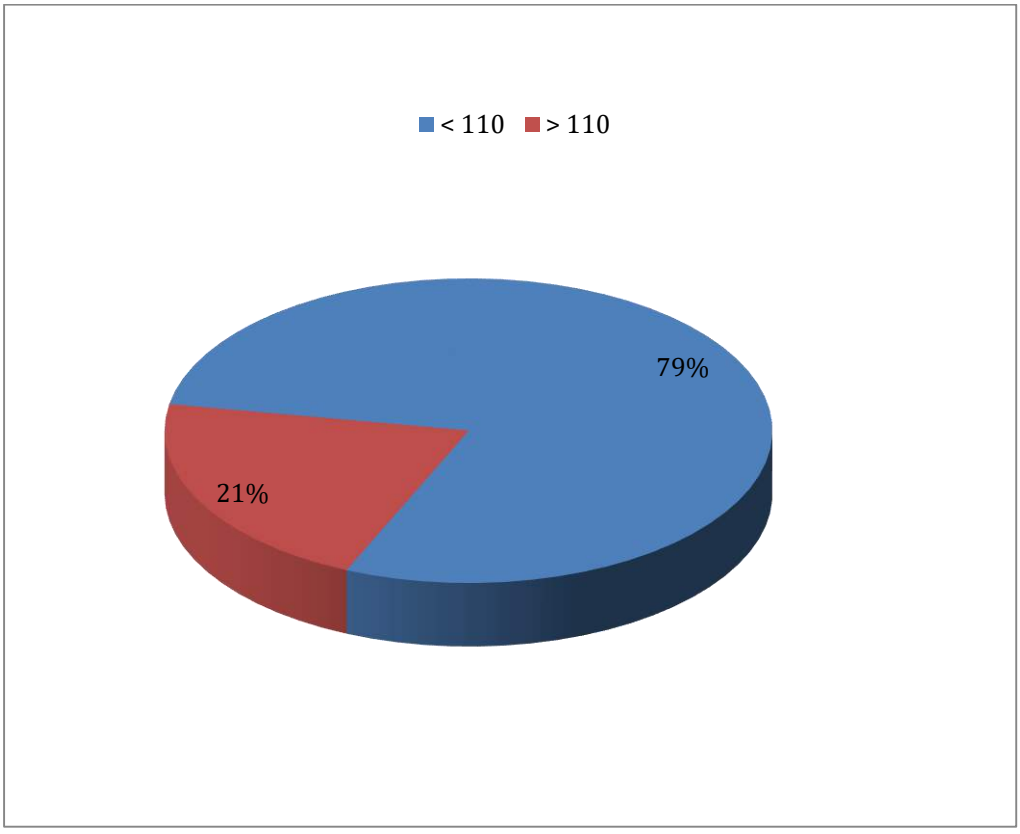

FIGURE 7: FBS IN PATIENTS

FREQUENCY OF VARIOUS DERMATOSES:

In our study which included 250 cases there were many patients with multiple cutaneous manifestations, so in total there were 354 dermatoses.

- Maximum number was seen between 41-50 years of age with 108 dermatoses (30.51%) and minimum was seen in age group of >70years with 9 dermatoses (2.54%).
- Acrochordons was the most common dermatoses in our study which was present in 87 cases (24.58%), followed by XP in 40 (11.30%) and folliculitis in 35 (9.89%) cases.

TABLE 8: AGE DISTRIBUTION ACCORDING TO DERMATOSES

Age (Years) GROUP	No. of cases acc to dermatoses	Percentage
18-20.0	11	3.11%
21-30	32	9.04%
31-40	100	28.25%
41-50	108	30.51%
51-60	64	18.08%
61-70	30	8.47%
>70	9	2.54%

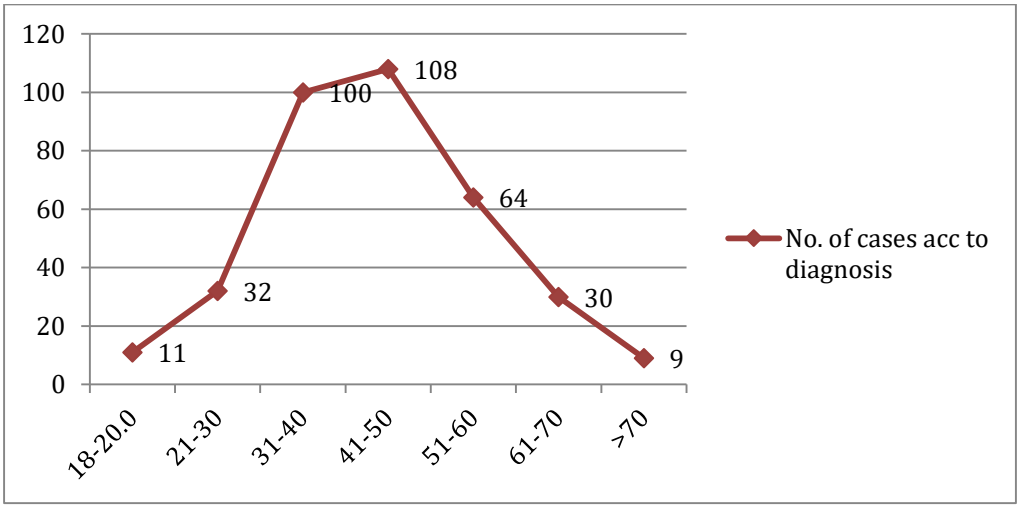

FIGURE 8: AGE DISTRIBUTION ACCORDING TO DERMATOSES

In this study which included 250 cases there were many patients with multiple cutaneous manifestations, so in total there were 354 dermatoses, 182 out of 354(51.41%) dermatoses were present in females

TABLE 9: GENDER DISTRIBUTION ACCORDING TO DERMATOSES

Gender	No. of cases acc to diagnosis	Percentage
F	182	51.41%
M	172	48.59%

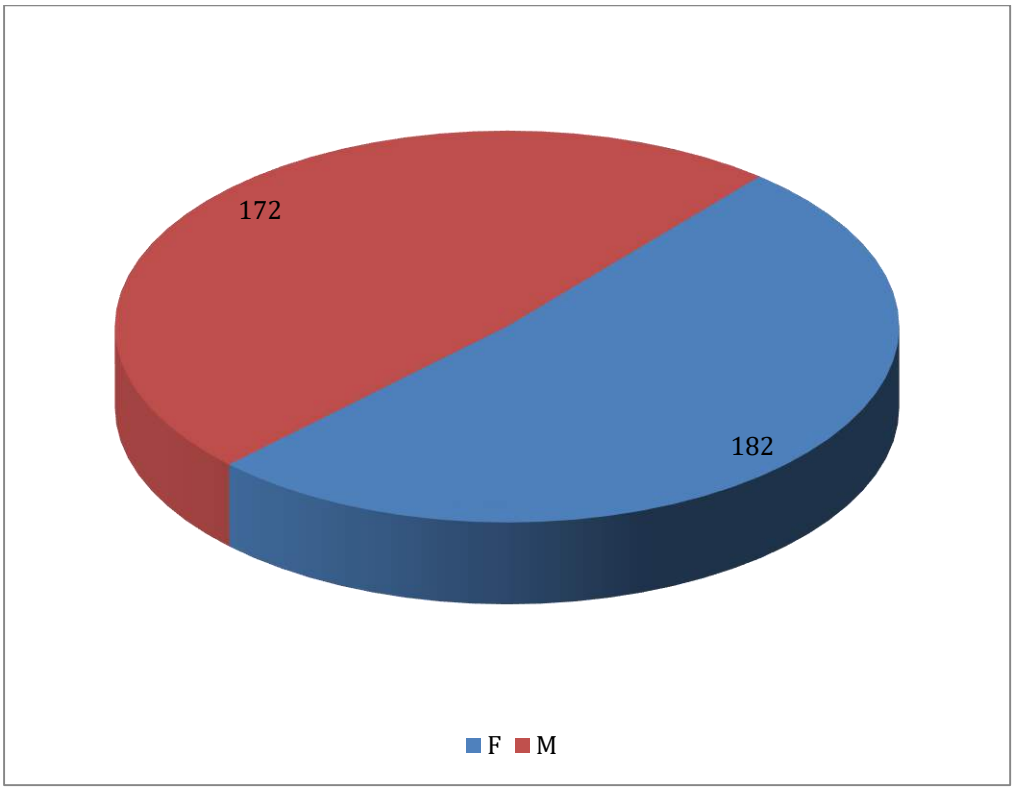

FIGURE9: GENDER DISTRIBUTION ACCORDING TO DERMATOSES

Distribution of dermatoses according to BMI, WC and WHR

- Out of 354 dermatoses, 205 (57.91%) were in grade I obesity and 149(42.09%) in grade II obesity.
- WC was higher in 325(91.81%) dermatoses and WHR was higher in 264 (74.57%) cases.

TABLE 10: DISTRIBUTION OF DERMATOSES ACCORDING TO BMI, WC AND WHR

	No. of cases acc. to dermatoses	Percentage
BMI GROUP		
I	205	57.91%
II	149	42.09%
Waist circumference(cm)		
Normal	29	8.19%
High	325	91.81%
Waist-hip ratio		
Normal	90	25.71%
High	264	74.29%

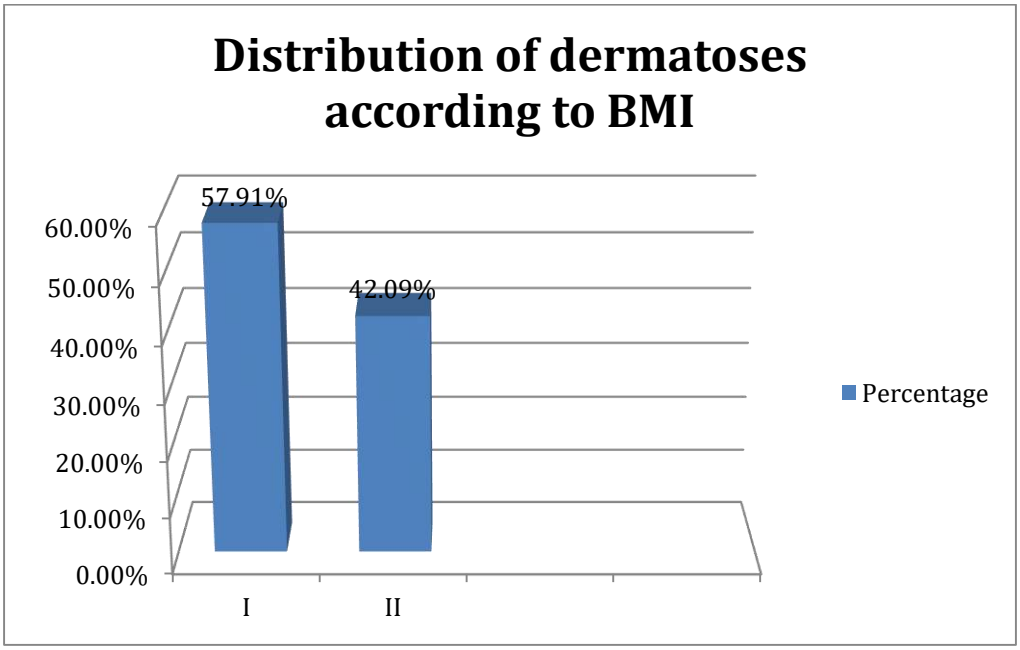

FIGURE 10: DISTRIBUTION OF DERMATOSES ACCORDING TO BMI

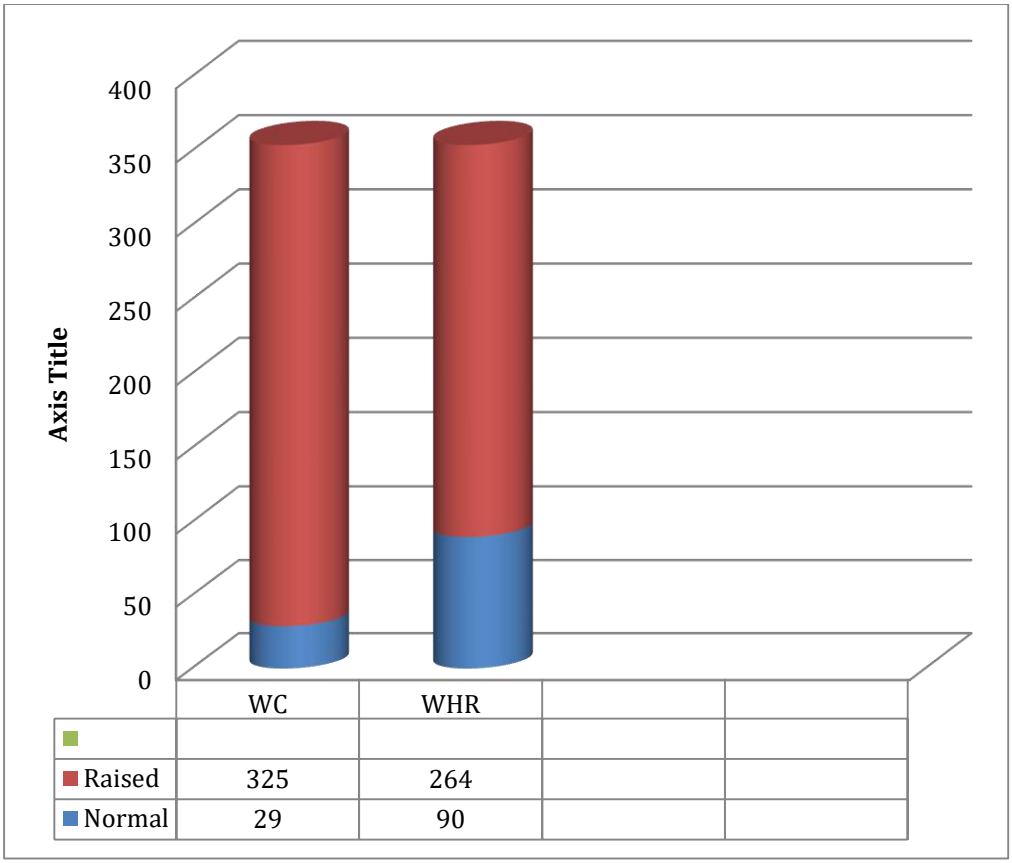

FIGURE 11: DISTRIBUTION OF DERMATOSES ACCORDING TO WC AND WHR

TABLE 11: FREQUENCY OF INDIVIDUAL DERMATOSES

	No. of cases	Percentage
ACROCHORDONS	87	24.58%
XANTHELASMA PALPEBRUM (XP)	40	11.30%
FOLLICULITIS	35	9.89%
INTERTRIGO	34	9.60%
TINEA CRURIS	32	9.04%
TINEA CORPORIS	30	8.47%
ACANTHOSIS NIGRICANS (AN)	24	6.78%
PYODERMA	17	4.80%
STRIAE DISTENSAE	16	4.52%
BALANOPOSTHITIS	4	1.13%
CORNS	4	1.13%
ONYCHOMYCOSIS	4	1.13%
PSORIASIS VULGARIS	6	1.69%
CELLULITIS	2	0.56%
CHRONIC PARONYCHIA	2	0.56%
GENERALISED PRURITUS	2	0.56%
ORAL THRUSH	3	0.85%
SEBORRHOEIC DERMATITIS	2	0.56%
ACUTE PARONYCHIA	1	0.28%
ASTEOTIC ECZEMA	1	0.28%
HERPES GENITALIS	1	0.28%
HERPES ZOSTER	1	0.28%
HIDRADENITIS SUPPURATIVA (HS)	1	0.28%
MOLLUSCUM CONTAGIOSUM	1	0.28%
NON HEALING ULCER	1	0.28%
PLANE WARTS	1	0.28%
SCROTAL DERMATITIS	1	0.28%
VULVO-VAGINAL CANDIDIASIS (VVC)	1	0.28%

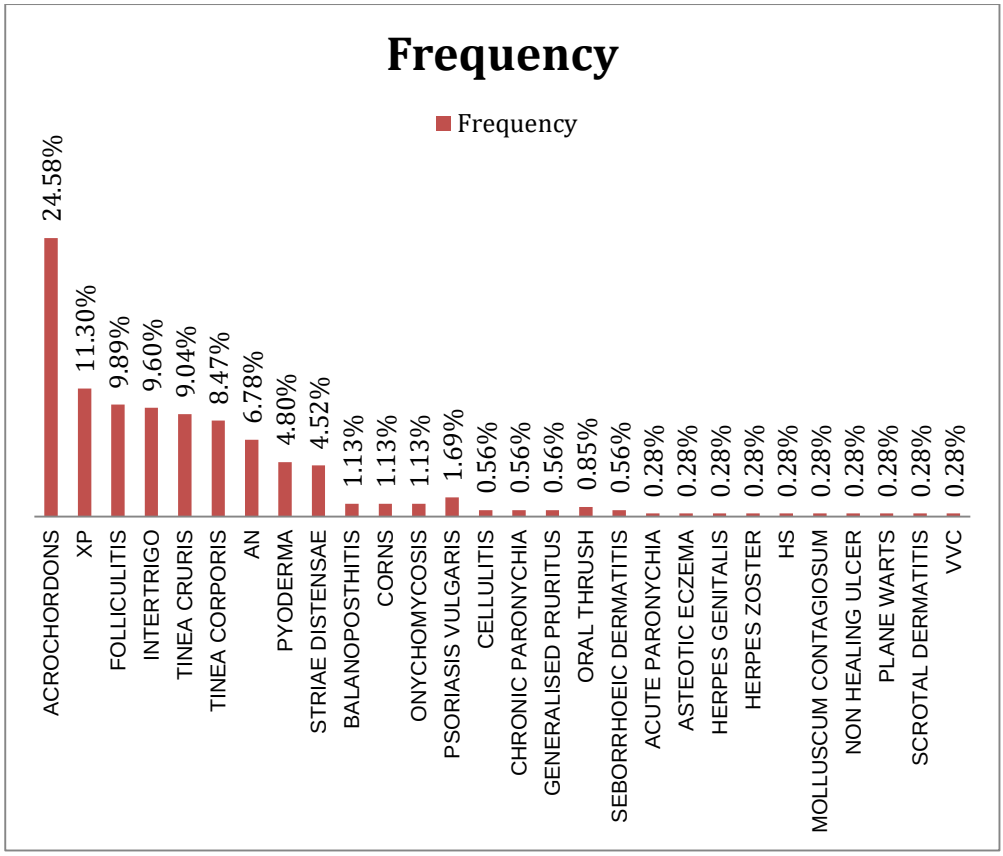

FIGURE 12: FREQUENCY OF INDIVIDUAL DERMATOSES

AGE-WISE DISTRIBUTION OF COMMON DERMATOSES:

Distribution of individual dermatoses in different age groups was variable such as acrochordons which was most common in our study was more in age group of 41-50 followed by 31-40; XP was more in 31-40 years age group followed by 41-50 years age group, with minimum number of cases in 18-20 and 20-30 years respectively.

TABLE 12:AGE-WISE DISTRIBUTION OF COMMON DERMATOSES

Age group	Acrochordons	XP	Folliculitis	Intertrigo	Tinea corporis	Tinea cruris	AN	Pyoderma	Striae	Psoriasis
18-20.0	1	0	2	0	1	1	2	0	0	0
21-30	2	2	10	2	4	3	1	3	1	0
31-40	24	14	6	6	11	11	9	5	6	3
41-50	31	13	7	14	5	9	7	4	9	1
51-60	21	8	8	5	6	4	3	1	0	2
61-70	8	3	1	6	3	3	1	3	0	0
>70	0	0	1	1	0	1	1	1	0	0

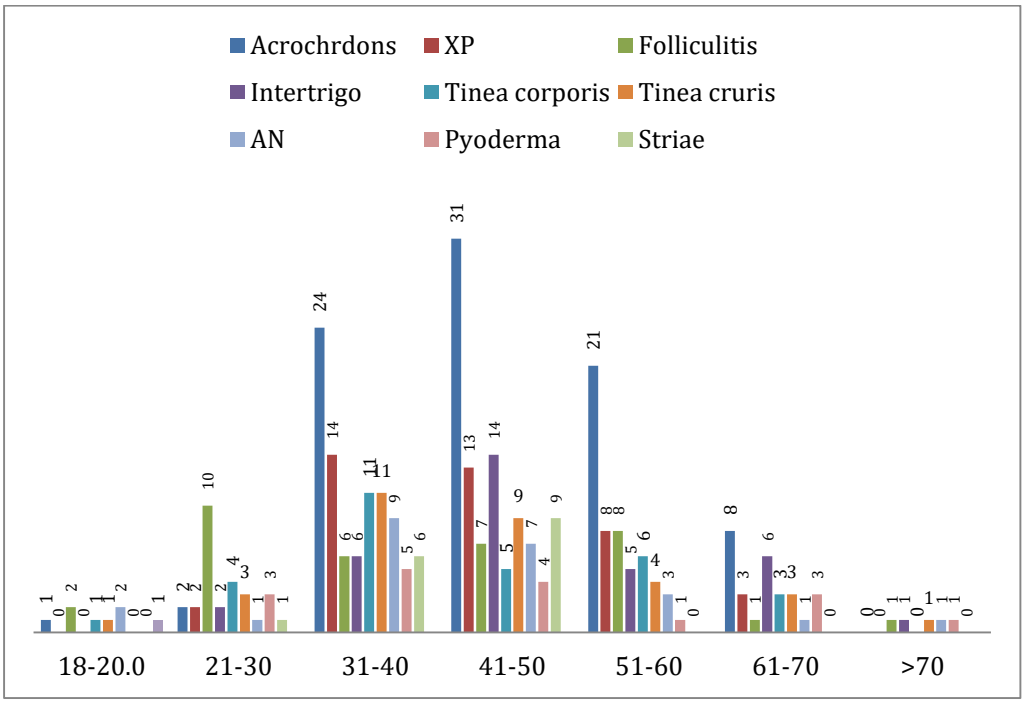

FIGURE 13:AGE-WISE DISTRIBUTION OF COMMON DERMATOSES

GENDER DISTRIBUTION OF DERMATOSES:

Gender wise distribution was variable among various dermatoses (Table 13). A statistical significant distribution was seen in females with XP (p-value 0.0001), males with tinea cruris (p-value 0.043) and AN (p-value 0.002).

TABLE 13: GENDER DISTRIBUTION OF INDIVIDUAL DERMATOSES

	Male(n)	Male(%)	Female(n)	Female(%)
ACROCHORDONS	41	47.13	46	52.87
XP	6	15	34	85
FOLLICULITIS	15	42.86	20	57.14
INTERTRIGO	14	41.18	20	58.82
TINEA CRURIS	21	65.62	11	34.38
TINEA CORPORIS	16	53.33	14	46.67
AN	19	79.17	5	20.83
PYODERMA	11	64.71	6	35.29
STRIAE DISTENSAE	7	43.75	9	56.25
BALANOPOSTHITIS	4	100	0	0
CORNS	1	25	3	75
ONYCHOMYCOSIS	2	50	2	50
PSORIASIS VULGARIS	4	66.7	2	33.3
CELLULITIS	1	50	1	50
CHRONIC PARONYCHIA	1	50	1	50
GENERALISED PRURITUS	2	100	0	0
ORAL THRUSH	1	33.3	2	66.7
SEBORRHOEIC DERMATITIS	1	50	1	50
ACUTE PARONYCHIA	0	0	1	100
ASTEOTIC ECZEMA	1	100	0	0
HERPES GENITALIS	1	100	0	0
HERPES ZOSTER	1	100	0	0
HS	1	100	0	0
MOLLUSCUM CONTAGIOSUM	0	0	1	100

NON HEALING ULCER	0	0	1	100
PLANE WARTS	0	0	1	100
SCROTAL DERMATITIS	1	100	0	0
VVC	0	0	1	100

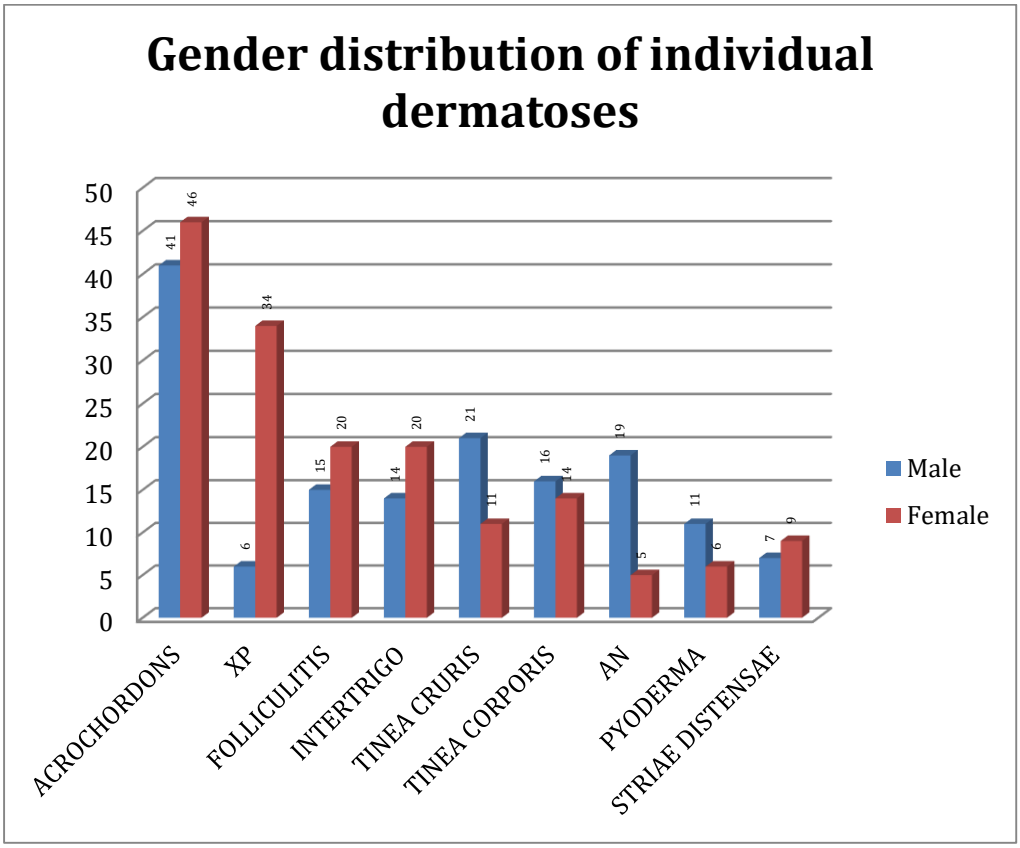

FIGURE 14: GENDER DISTRIBUTION OF INDIVIDUAL DERMATOSES

ASSOCIATION OF DERMATOSES WITH GRADE OF OBESITY:

Table 14 and figure 15 shows distribution of individual dermatoses according to grade of obesity.

Some cases showed statistically significant difference in distribution among various grades of obesity as in case of AN with 75% cases in grade II obesity, striae with 93.7% cases in grade II obesity, pyoderma with 100% cases in grade I obesity and folliculitis with 82.8% cases in grade I obesity with a p-value of 0.001, 0.0001, 0.001 and 0.001 respectively.

TABLE 14: DISTRIBUTION OF INDIVIDUAL DERMATOSES ACCORDING TO GRADE OF OBESITY

	GRADE I	GRADE II
ACROCHORDONS	44	43
XANTHELASMA PALPEBRUM	21	19
FOLLICULITIS	29	6
INTERTRIGO	20	14
TINEA CRURIS	20	12
TINEA CORPORIS	19	11
ACANTHOSIS NIGRICANS	6	18
PYODERMA	17	0
STRIAE DISTENSAE	1	15
BALANOPOSTHITIS	4	0
CORNS	2	2
ONYCHOMYCOSIS	2	2
PSORIASIS VULGARIS	5	1
CELLULITIS	2	0
CHRONIC PARONYCHIA	1	1
GENERALISED PRURITUS	2	0
ORAL THRUSH	2	1
SEBORRHOEIC DERMATITIS	2	0
ACUTE PARONYCHIA	0	1
ASTEOTIC ECZEMA	1	0

HERPES GENITALIS	1	0
HERPES ZOSTER	1	0
HIDRADENITIS SUPPURATIVA	1	0
MOLLUSCUM CONTAGIOSUM	0	1
NON HEALING ULCER	0	1
PLANE WARTS	0	1
SCROTAL DERMATITIS	1	0
VULVOVAGINAL CANDIDIASIS	1	0
Total	205	149

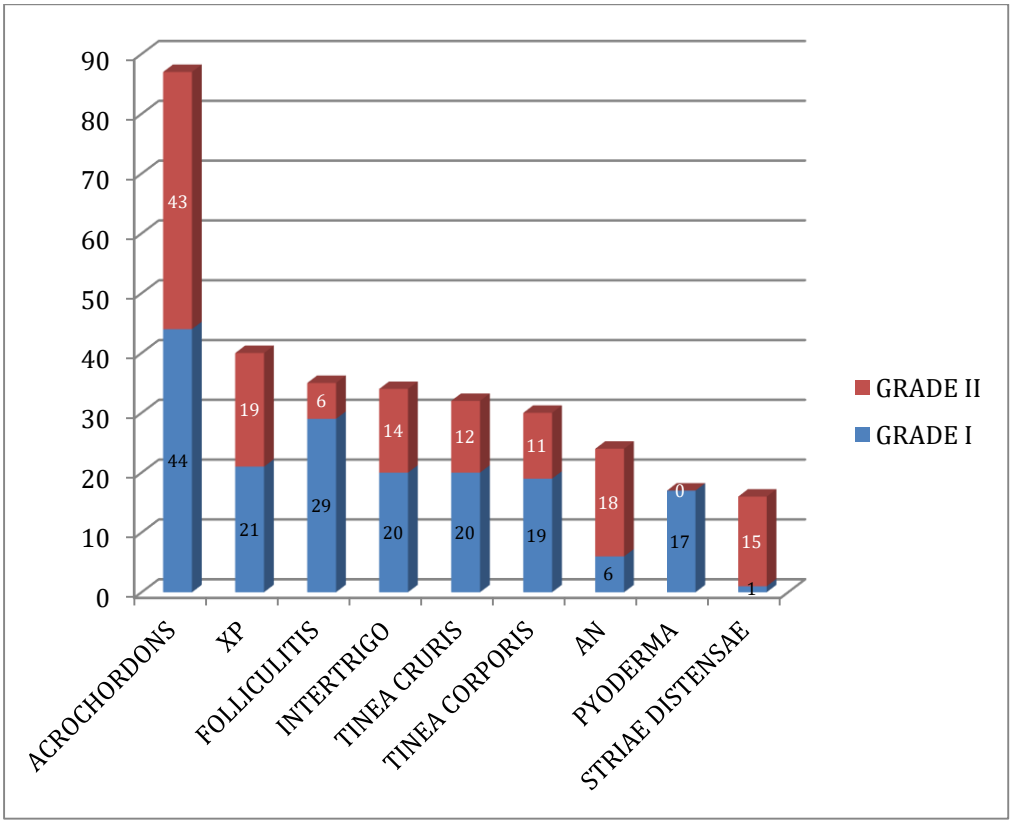

FIGURE 15: MAJOR DISEASES IN THE STUDY AND THEIR ASSOCIATION WITH GRADES OF OBESITY

ASSOCIATION OF DERMATOSES WITH WC AND WHR:

Table 15 and figure 16 shows distribution of various dermatoses according to anthropometric measurements.

- WC was raised in all cases of striae distensae and XP. WC was raised in a higher number of cases in acrochordons (86/87), XP(40/40), intertrigo (33/34). Statistical significant increase of WC was seen in acrochordons (p-value 0.019), pyoderma (p-value 0.030).
- WHR was increased in 100% cases of striae distensae. WHR was higher in cases with acrochordons (69/87), XP (37/40), intertrigo (26/34), AN (15/24), psoriasis (5/6). WHR was statistically significant in XP (p-value 0.011), pyoderma (p-value 0.002), striae (p-value 0.015)

TABLE 15: ASSOCIATION OF INDIVIDUAL DERMATOSES WITH WC AND WHR:

Dermatoses	Waist circumference		Waist-Hip Ratio	
	Normal	High	Normal	High
ACROCHORDONS	1	86	18	69
XP	0	40	3	37
FOLLICULITIS	5	30	11	24
INTERTRIGO	1	33	8	26
TINEA CRURIS	4	28	10	22
TINEA CORPORIS	4	26	9	21
AN	1	23	9	15
PYODERMA	4	13	10	7
STRIAE DISTENSAE	0	16	0	16
BALANOPOSTHITIS	1	3	2	2
CORNS	1	3	0	4
ONYCHOMYCOSIS	0	4	0	4
PSORIASIS VULGARIS	0	6	1	5
CELLULITIS	1	1	0	2
CHRONIC PARONYCHIA	0	2	1	1

GENERALISED PRURITUS	0	2	2	0
ORAL THRUSH	0	3	1	2
SEBORRHOEIC DERMATITIS	1	1	1	1
ACUTE PARONYCHIA	1	0	0	1
ASTEOTIC ECZEMA	1	0	0	1
HERPES GENITALIS	1	0	1	0
HERPES ZOSTER	1	0	1	0
HS	1	0	1	0
MOLLUSCUM CONTAGIOSUM	0	1	0	1
NON HEALING ULCER	0	1	0	1
PLANE WARTS	0	1	0	1
SCROTAL DERMATITIS	0	1	1	0
VVC	0	1	0	1

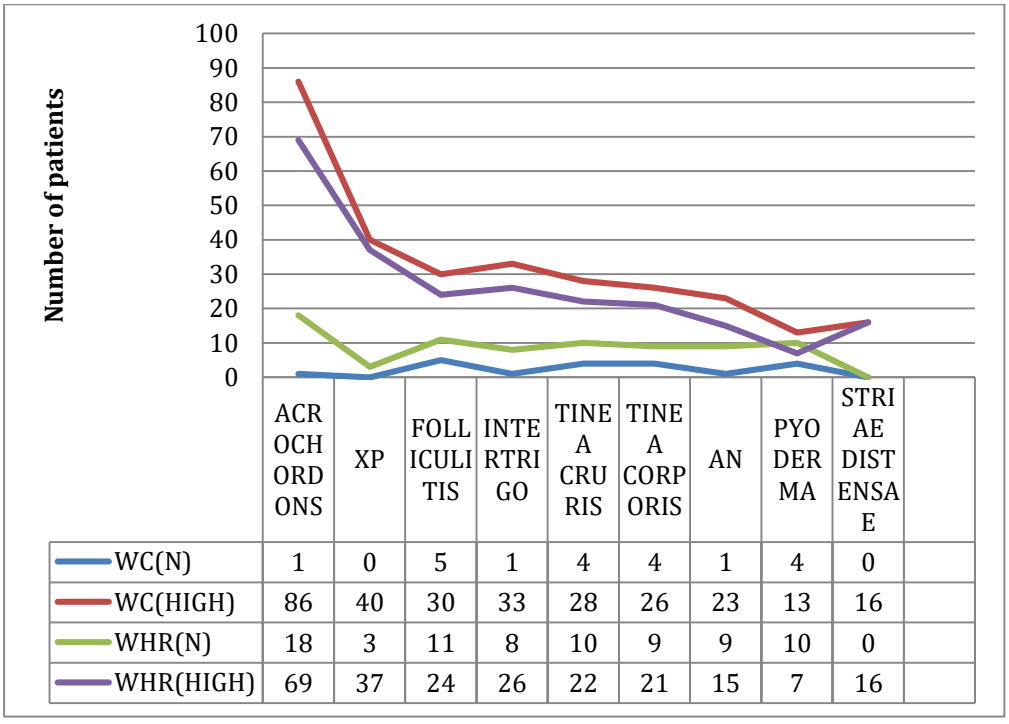

FIGURE 16: ASSOCIATION OF COMMON DERMATOSES WITH WC AND WHR

ASSOCIATION OF INDIVIDUAL DERMATOSES WITH LIPID PROFILE AND FBS:

Table 16 and figure 17 shows various lab changes in individual dermatoses.

- Striae distensae was frequently associated with raised total cholesterol levels with 62.50% cases followed by XP (45%) and AN (41.67%).
- FBS was raised in balanoposthitis (75%), 41.18% cases of intertrigo and 37.50% cases of striae distensae. A statistical significant change in FBS was seen in intertrigo with p-value 0.026.
- A statistical significant change in TC and TG was seen in striae distensae with p-value 0.025 and 0.041 respectively. A statistical significant change in TC was seen in pyoderma with p-value 0.0001. A statistical significant change in TG was seen in acrochordons and folliculitis with p-value 0.048 and 0.004 respectively.

TABLE16: LAB CHANGES IN INDIVIDUAL DISEASES

	TC<200	TC>200	TG<200	TG>200	FBS<110	FBS>110
ACROCHORDONS	52	35	51	36	60	27
XANTHELASMA PALPEBRUM	22	18	23	17	34	6
FOLLICULITIS	27	8	31	4	29	6
INTERTRIGO	21	13	26	8	20	14
TINEA CRURIS	21	11	22	10	23	9
TINEA CORPORIS	18	12	22	8	25	5
ACANTHOSIS NIGRICANS	14	10	12	12	18	6
PYODERMA	13	4	13	4	15	2
STRIAE DISTENSAE	6	10	7	9	10	6

BALANOPOSTHITIS	3	1	3	1	1	3
CORNS	3	1	4	0	3	1
ONYCHOMYCOSIS	3	1	2	2	3	1
PSORIASIS VULGARIS	4	2	5	1	6	0
CELLULITIS	2	0	2	0	1	1
CHRONIC PARONYCHIA	2	0	2	0	2	0
GENERALISED PRURITUS	2	0	1	1	2	0
ORAL THRUSH	2	1	1	2	2	1
SEBORRHOEIC DERMATITIS	2	0	2	0	2	0
ACUTE PARONYCHIA	1	0	1	0	1	0
ASTEOTIC ECZEMA	1	0	1	0	0	1
HERPES GENITALIS	1	0	1	0	1	0
HERPES ZOSTER	1	0	1	0	1	0
HIDRADENITIS SUPPURATIVA	1	0	1	0	1	0
MOLLUSCUM CONTAGIOSUM	1	0	1	0	1	0
NON-HEALING ULCER	1	0	1	0	0	1
PLANE WARTS	0	1	0	1	1	0
SCROTAL DERMATITIS	1	0	1	0	1	0
VULVOVAGINAL CANDIDIASIS	1	0	1	0	1	0

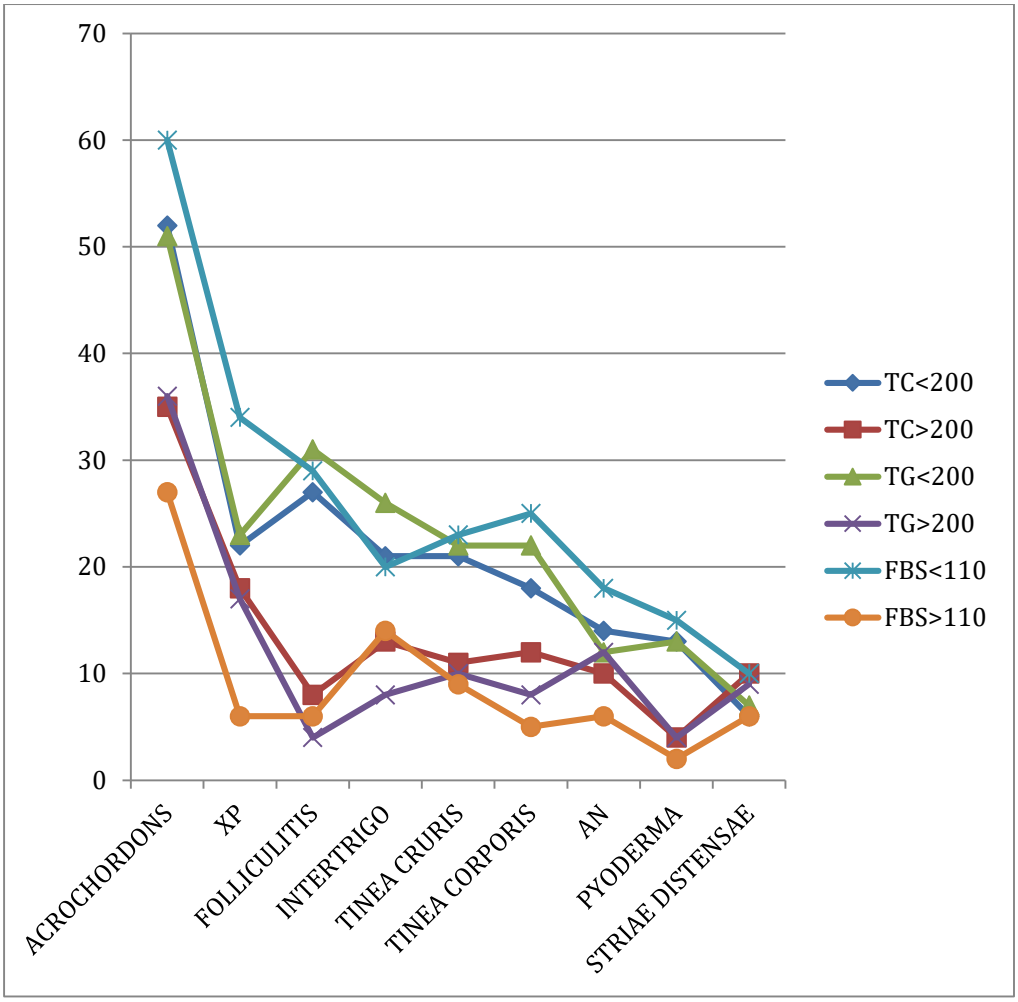

FIGURE 17: LAB CHANGES IN INDIVIDUAL DISEASES

ACROCHORDONS:

- Skin tags were the most common dermatological manifestation in our study were present in 87 cases, more common in age-group of 41-50 years with 31 cases.
- There was slight female preponderance with 46 (52.87%) cases in females compared to 41(47.13%) cases in male patients. Out of total 87 patients 26 were diabetic, 44 cases in grade I obesity and 43 in grade II.
- WC and WHR were raised in 86 and 69 cases respectively and TC, TG, LDL were increased in 35, 36, 36 cases respectively.
- Multiple sites were involved in several patients, neck was most commonly involved with 87 cases followed by axilla in 13 cases; most common association was with AN(15) followed by striae, XP (13 cases both).

TABLE 17: GENDER DISTRIBUTION OF PATIENTS WITH ACROCHORDONS

Male(n)	Male(%)	Female(n)	Female(%)
41	47.13	46	52.87

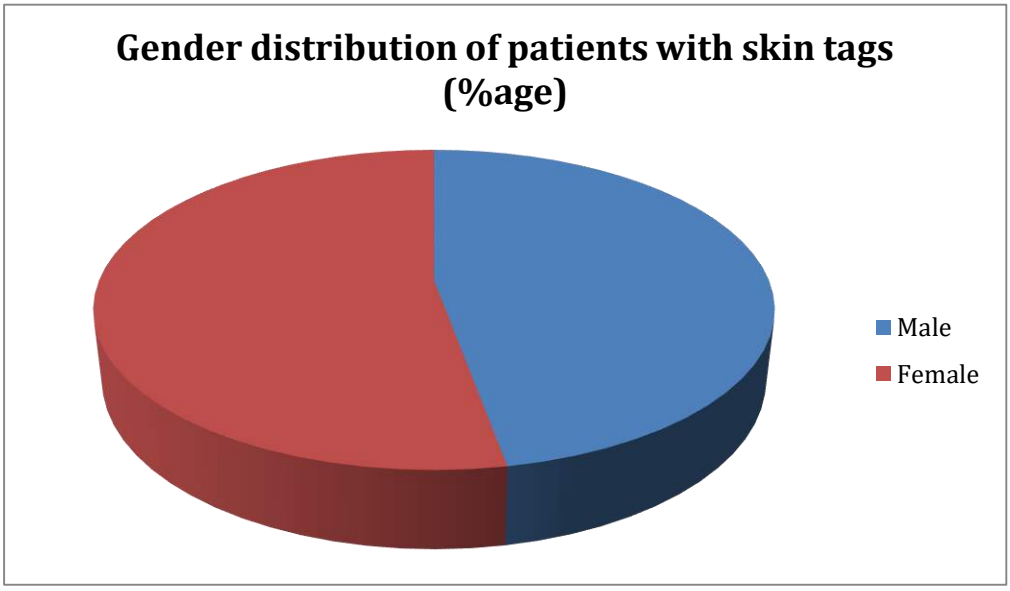

FIGURE 18: GENDER DISTRIBUTION OF PATIENTS WITH ACROCHORDONS (%AGE)

TABLE 18: DISTRIBUTION OF PATIENTS WITH ACROCHORDONS ACCORDING TO WC AND WHR

Dermatoses	Waist circumference		Waist-Hip Ratio	
	Normal	High	Normal	High
ACROCHORDONS	1	86	18	69

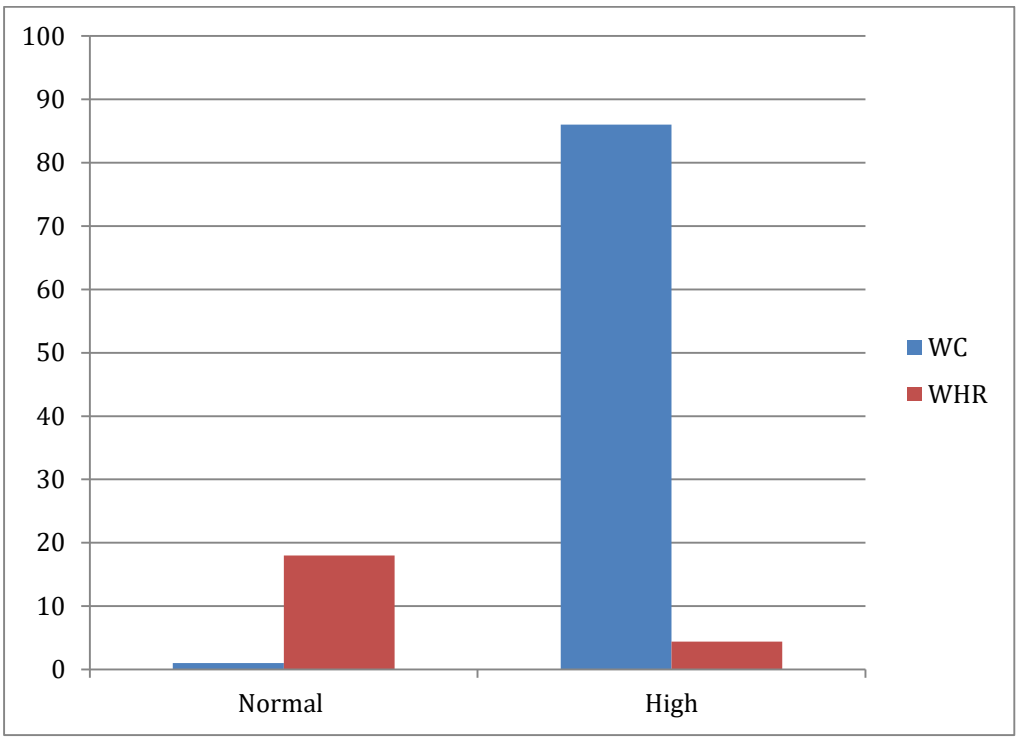

FIGURE 19: DISTRIBUTION OF PATIENTS WITH SKIN TAGS ACCORDING TO WC AND WHR

TABLE 19: DISTRIBUTION OF PATIENTS WITH SKIN TAGS ACCORDING TO GRADES OF OBESITY

	GRADE I	GRADE II
ACROCHORDONS	44	43

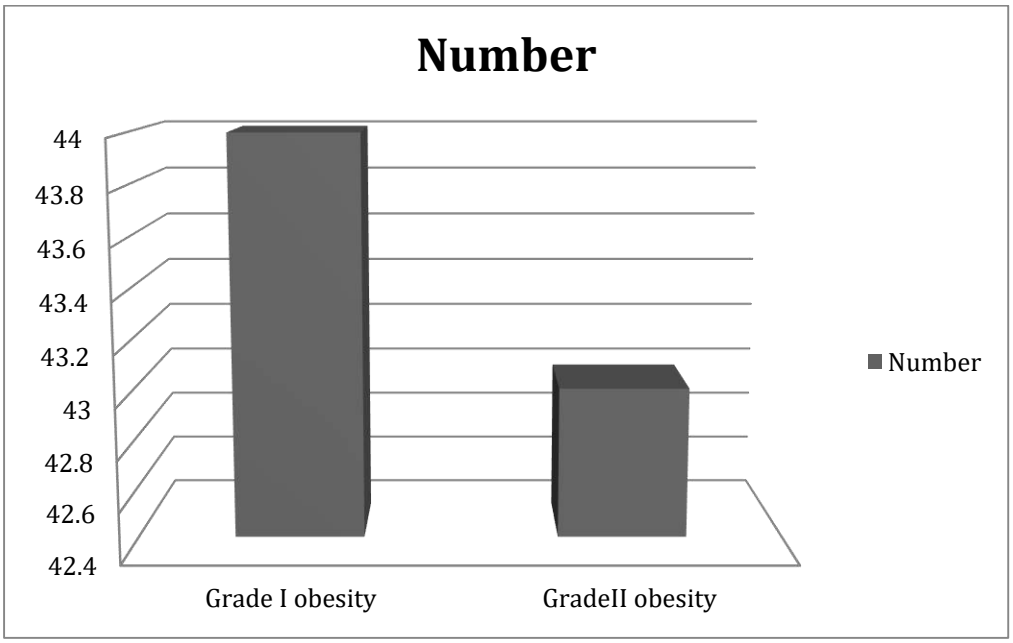

FIGURE 20: GRADING OF PATIENTS WITH ACROCHORDON BASED ON BMI

TABLE 20: FREQUENCY OF FBS AND LIPID PROFILE ALTERATIONS IN PATIENTS WITH ACROCHORDONS

	TC<200	TC>200	TG<200	TG>200	FBS<110	FBS>110
ACROCHORDONS	52	35	51	36	60	27

XANTHELASMA PALPEBRUM

- Xanthelasma palpebrum was second most common dermatoses in our study with 40 cases(11.30%), with female preponderance 34 cases (85%) in female patients compared to 6 cases(15%) in males.
- XP was more common in 31-40 years age group, 19 cases were in patients of grade II obesity and 21 cases of grade I obesity; WC and WHR was raised in 40 and 37 cases respectively.

TABLE 21: GENDER DISTRIBUTION OF PATIENTS WITH XP

	Male(n)	Male(%)	Female(n)	Female(%)
XP	6	15	34	85

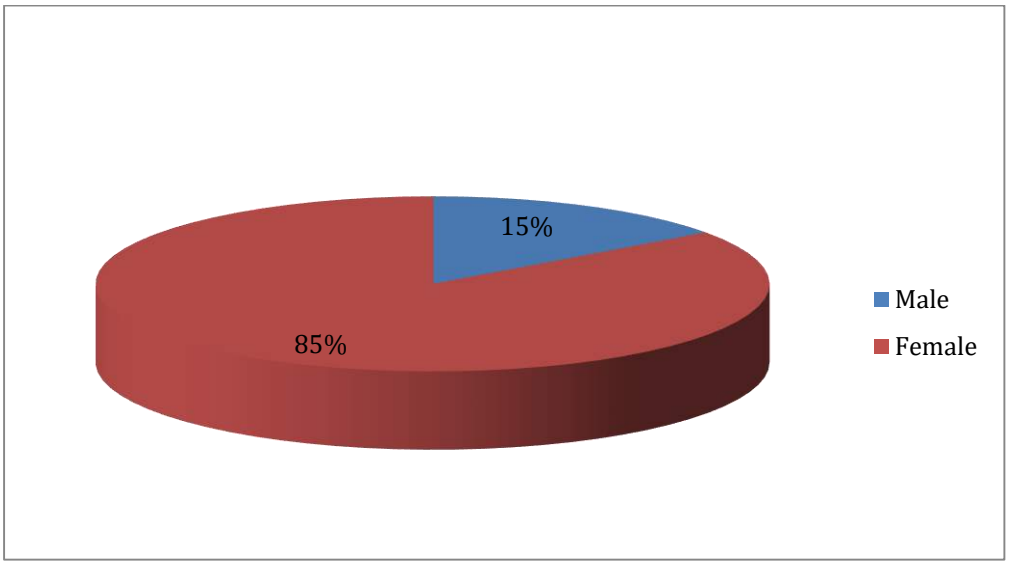

FIGURE 21: GENDER DISTRIBUTION OF PATIENTS WITH XP

TABLE 22: DISTRIBUTION OF PATIENTS WITH XP ACCORDING TO WC, WHR AND BMI

Dermatoses	Waist circumference		Waist-Hip Ratio		BMI	
	Normal	High	Normal	High	Grade I	Grade II
XP	0	40	3	37	21	19

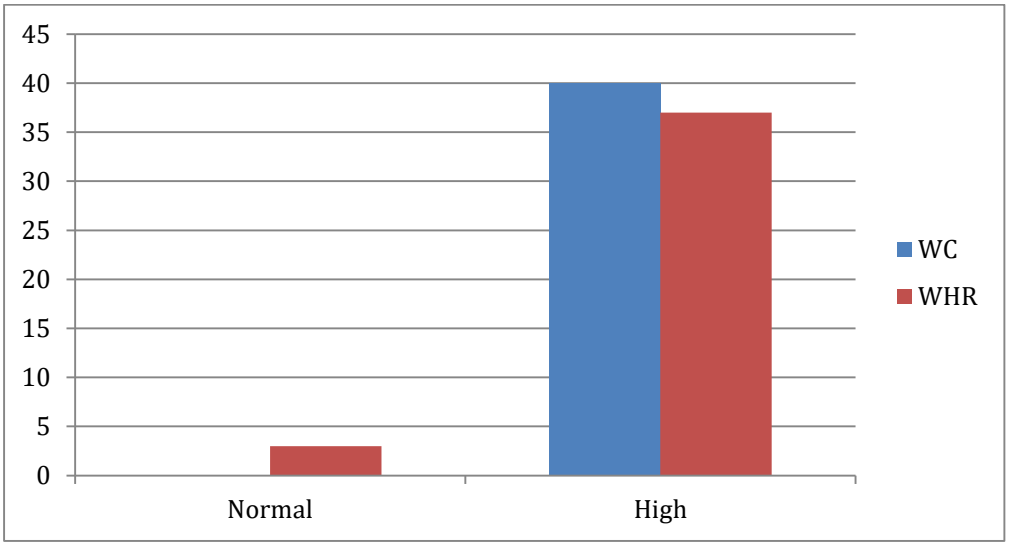

FIGURE 22: DISTRIBUTION OF PATIENTS WITH XP ACCORDING TO WC AND WHR

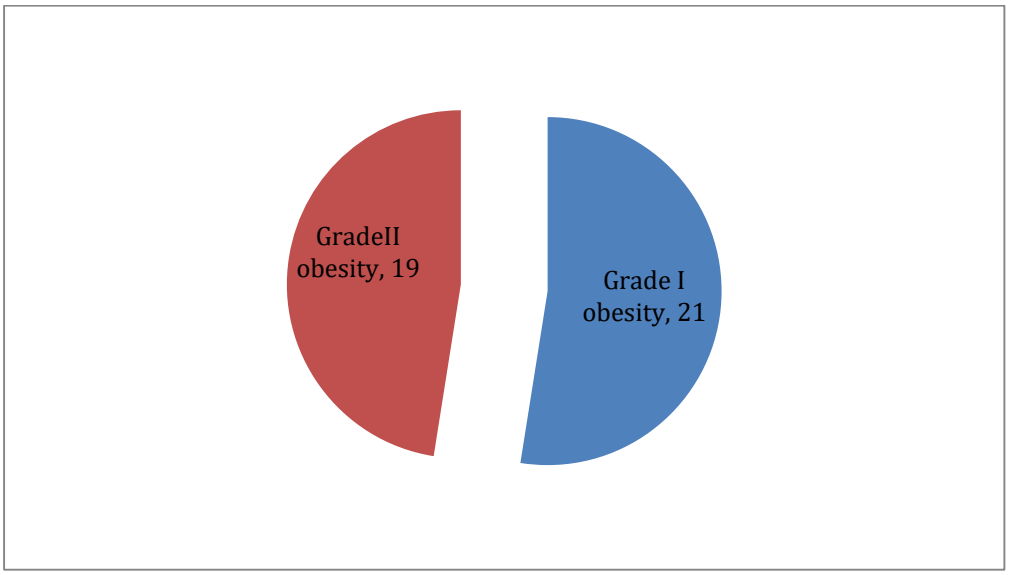

FIGURE 23: DISTRIBUTION OF PATIENTS WITH XP ACCORDING TO BMI

TABLE 23: FREQUENCY OF FBS AND LIPID PROFILE ALTERATIONS IN PATIENTS WITH XP

	TC<200	TC>200	TG<200	TG>200	FBS<110	FBS>110
XP	22	18	23	17	34	6

FOLLICULITIS

- In our study, there were 35 cases of folliculitis with no significant difference in gender based distribution.
- Most of cases were of grade I obesity with 29/35 cases as compared to 6 cases of grade II obesity with 85.7% cases having higher WC and 68.5% with a higher WHR.
- Only a few cases had lipid abnormalities as shown in table 26.

TABLE 24: GENDER DISTRIBUTION OF PATIENTS WITH FOLLICULITIS

	Male(n)	Male(%)	Female(n)	Female(%)
FOLLICULITIS	15	42.86	20	57.14

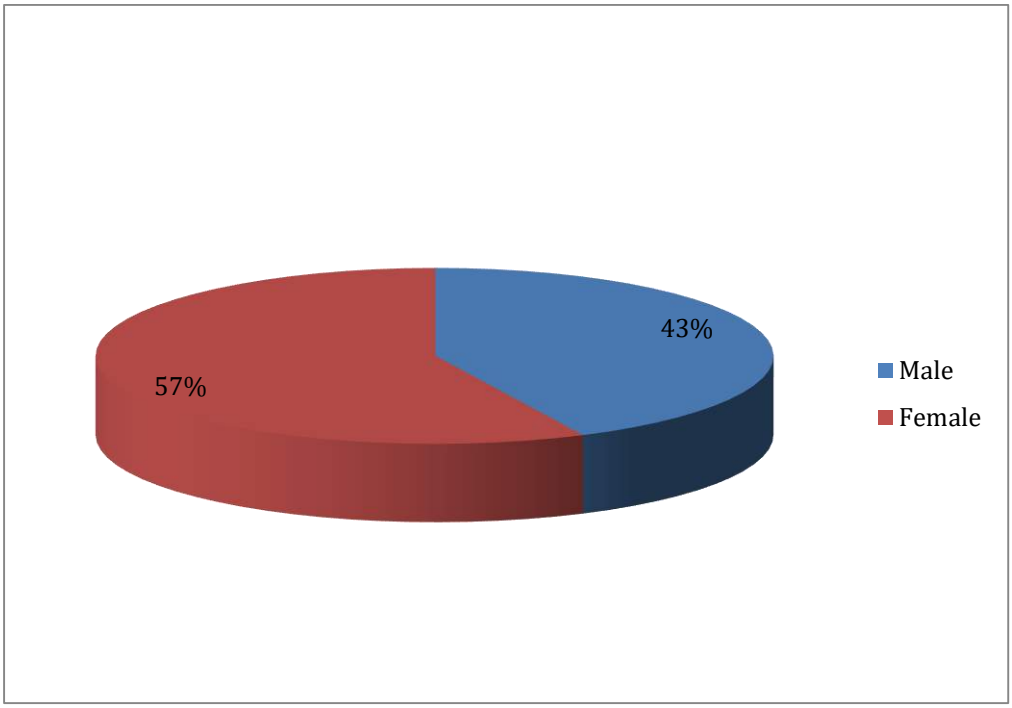

FIGURE 24: GENDER DISTRIBUTION OF PATIENTS WITH FOLLICULITIS

TABLE 25: DISTRIBUTION OF PATIENTS WITH FOLLICULITIS ACCORDING TO WC, WHR AND BMI

Dermatoses	Waist circumference		Waist-Hip Ratio		BMI	
	Normal	High	Normal	High	Grade I	Grade II
FOLLICULITIS	5	30	11	24	29	6

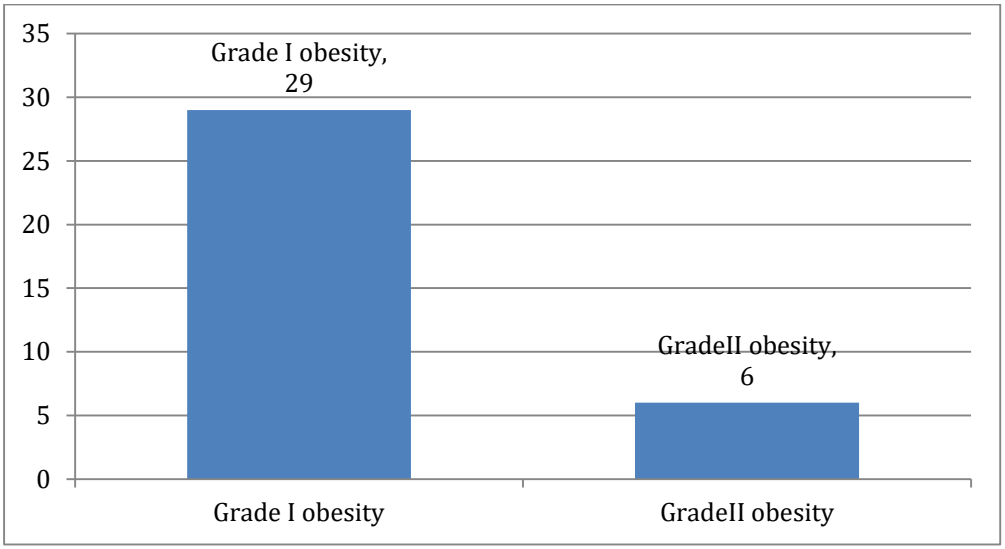

FIGURE 25: DISTRIBUTION OF PATIENTS WITH FOLLICULITIS ACCORDING TO BMI

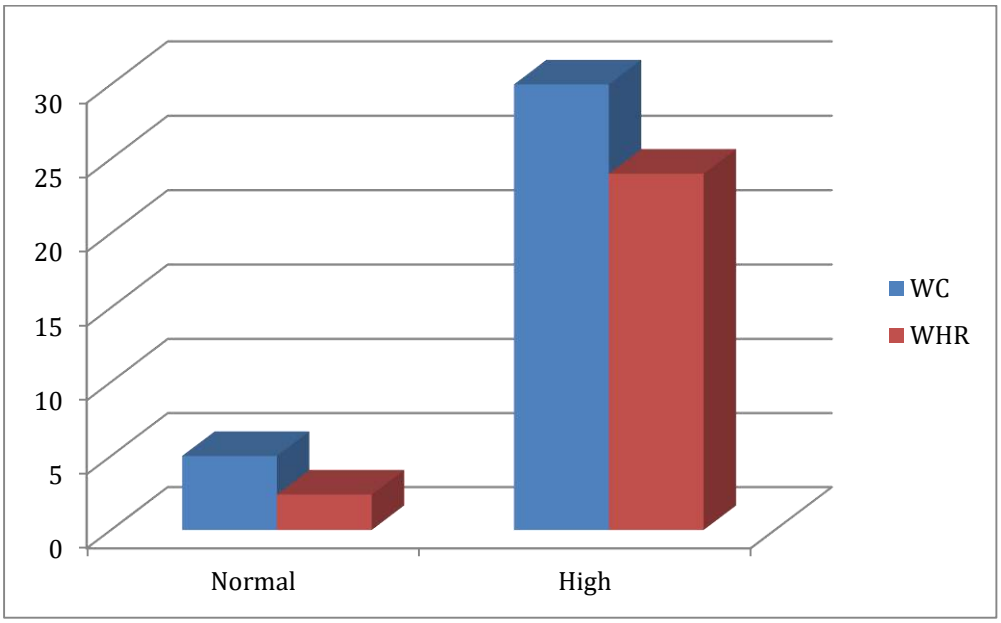

FIGURE 26: DISTRIBUTION OF PATIENTS WITH FOLLICULITIS ACCORDING TO WC AND WHR

INTERTRIGO

- In our study, 34 cases of intertrigo were seen with a slight female preponderance with 58.82%cases in females. 20 patients were of grade I obesity and 14 of grade II.
- WC and WHR were higher in 33 and 26 cases respectively and 14 patients had raised FBS, only 13 cases showed an increase in TC and 8 had raised TG levels.

TABLE 26: FREQUENCY OF FBS AND LIPID PROFILE ALTERATIONS IN PATIENTS WITH FOLLICULITIS

	TC<200	TC>200	TG<200	TG>200	FBS<110	FBS>110
FOLLICULITIS	27	8	31	4	29	6

TABLE 27: GENDER DISTRIBUTION OF PATIENTS WITH INTERTRIGO

	Male(n)	Male(%)	Female(n)	Female(%)
INTERTRIGO	14	41.18	20	58.82

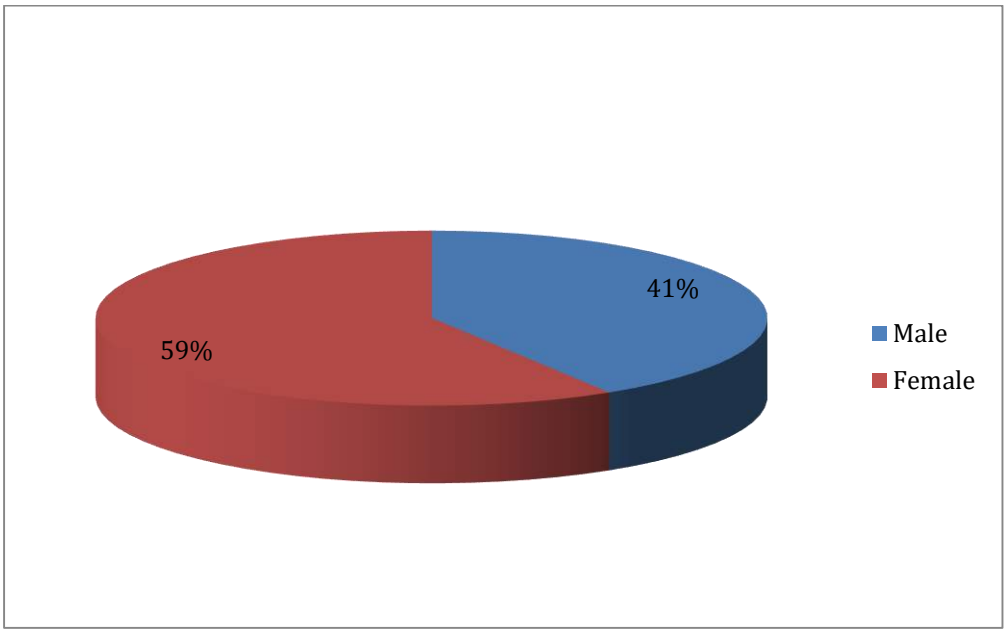

FIGURE 27: GENDER DISTRIBUTION OF PATIENTS WITH INTERTRIGO

TABLE 28: DISTRIBUTION OF PATIENTS WITH INTERTRIGO ACCORDING TO WHR,WC AND BMI

Dermatoses	Waist circumference		Waist-Hip Ratio		BMI	
	Normal	High	Normal	High	Grade I	Grade II
INTERTRIGO	1	33	8	26	20	14

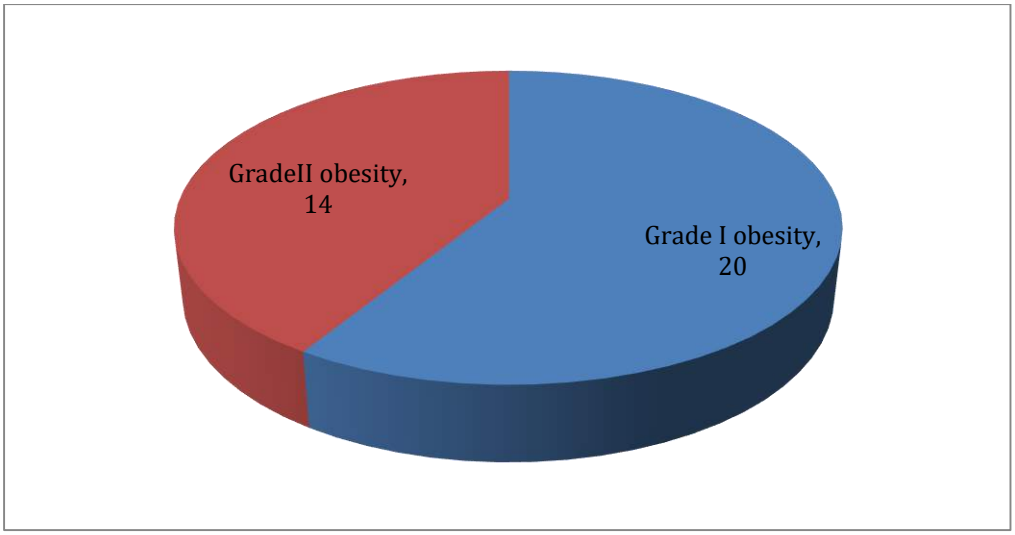

FIGURE 28: DISTRIBUTION OF PATIENTS WITH INTERTRIGO ACCORDING TO BMI

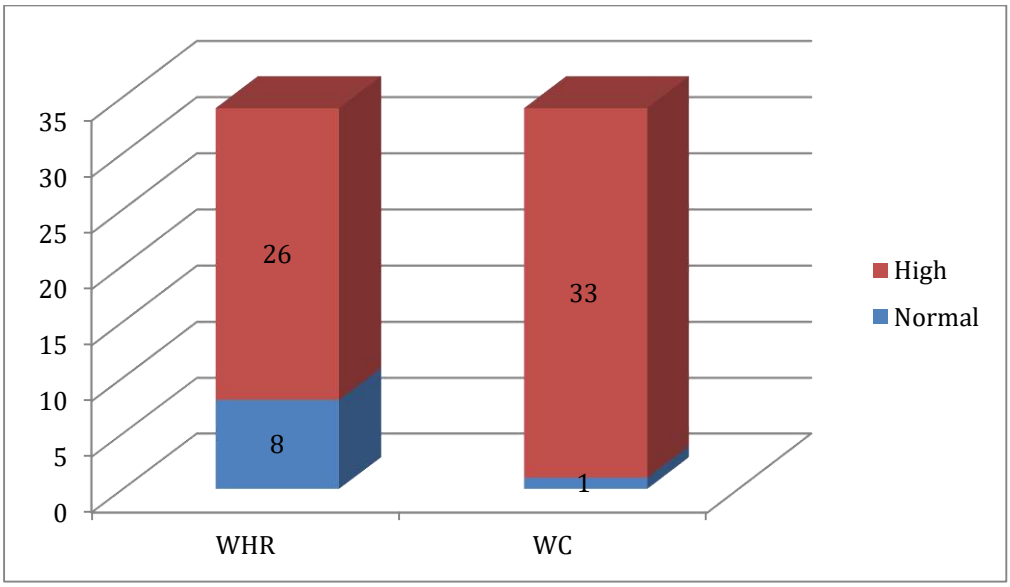

FIGURE 29: DISTRIBUTION OF PATIENTS WITH INTERTRIGO ACCORDING TO WC AND WHR

TABLE 29: FREQUENCY OF FBS AND LIPID PROFILE ALTERATIONS IN PATIENTS WITH INTERTRIGO

	TC<200	TC>200	TG<200	TG>200	FBS<110	FBS>110
INTERTRIGO	21	13	26	8	20	14

DERMATOPHYTIC INFECTIONS

- In our study there were 30 cases of tinea corporis, 32 cases of tinea cruris.
- There was slight male preponderance as shown in table 30.
- A significant proportion of cases had raised WC and WHR.

TABLE 30: GENDER DISTRIBUTION OF PATIENTS WITH DERMATOPHYTIC INFECTIONS

	Male(n)	Male(%)	Female(n)	Female(%)
TINEA CRURIS	21	65.62	11	34.38
TINEA CORPORIS	16	53.33	14	46.67

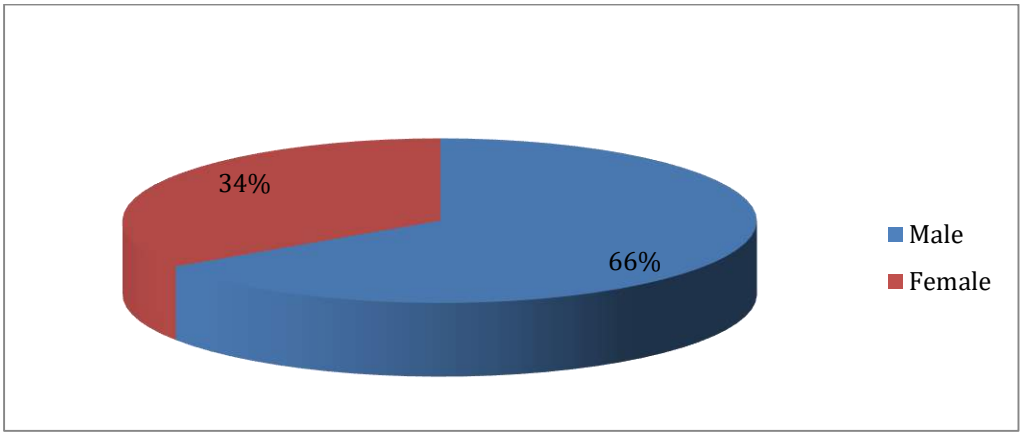

FIGURE 30: GENDER DISTRIBUTION OF PATIENTS WITH TINEA CRURIS

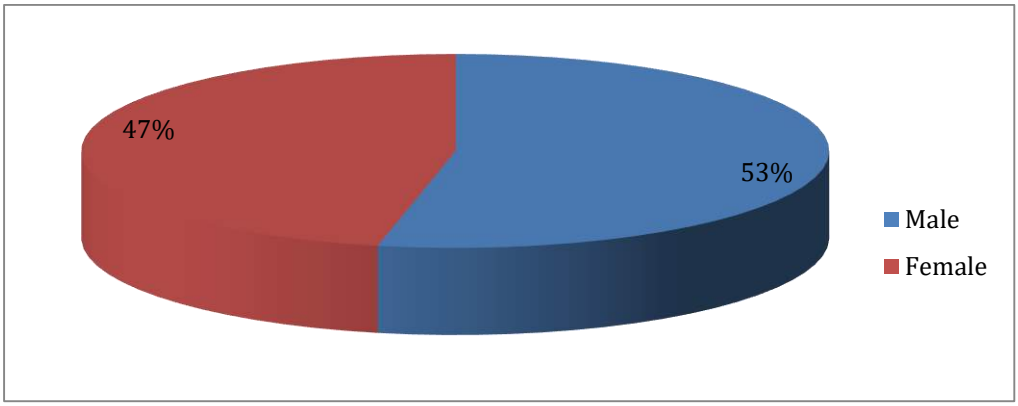

FIGURE 31: GENDER DISTRIBUTION OF PATIENTS WITH TINEA CORPORIS

TABLE 31: DISTRIBUTION OF PATIENTS WITH DERMATOPHYTIC INFECTIONS ACCORDING TO BMI

	GRADE I	GRADE II
TINEA CRURIS	20	12
TINEA CORPORIS	19	11

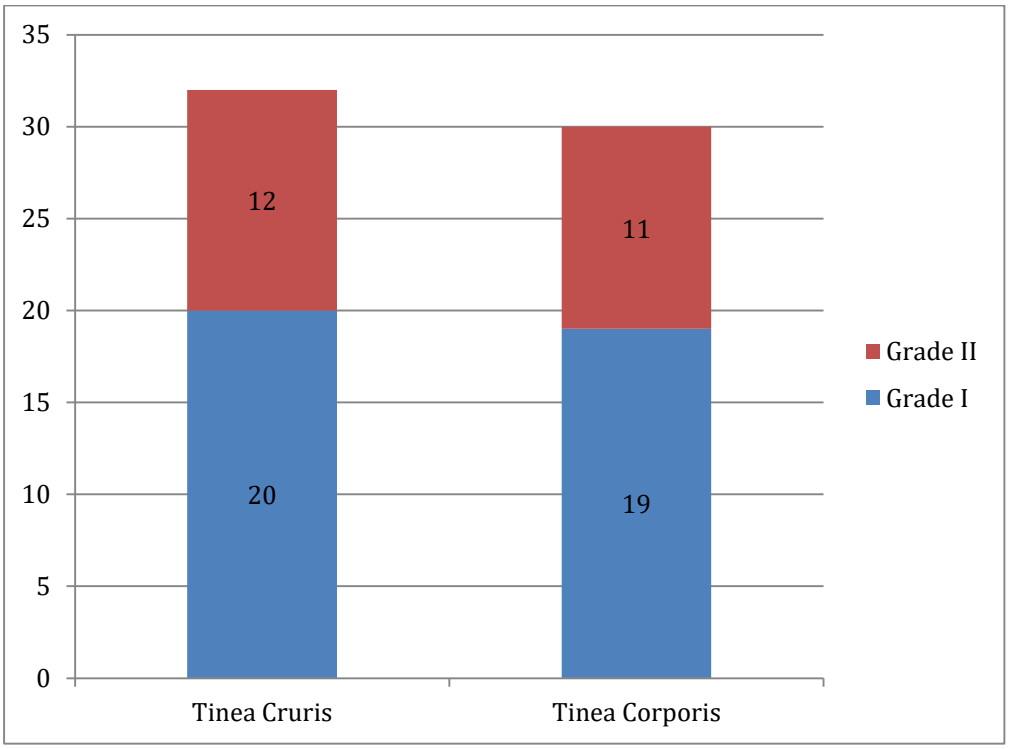

FIGURE 32: DISTRIBUTION OF PATIENTS WITH TINEA CRURIS AND TINEA CORPORIS ACCORDING TO BMI

TABLE 32: DISTRIBUTION OF PATIENTS WITH DERMATOPHYTIC INFECTIONS ACCORDING TO WC AND WHR

Dermatoses	Waist circumference		Waist-Hip Ratio	
	Normal	High	Normal	High
TINEA CRURIS	4	28	10	22
TINEA CORPORIS	4	26	9	21

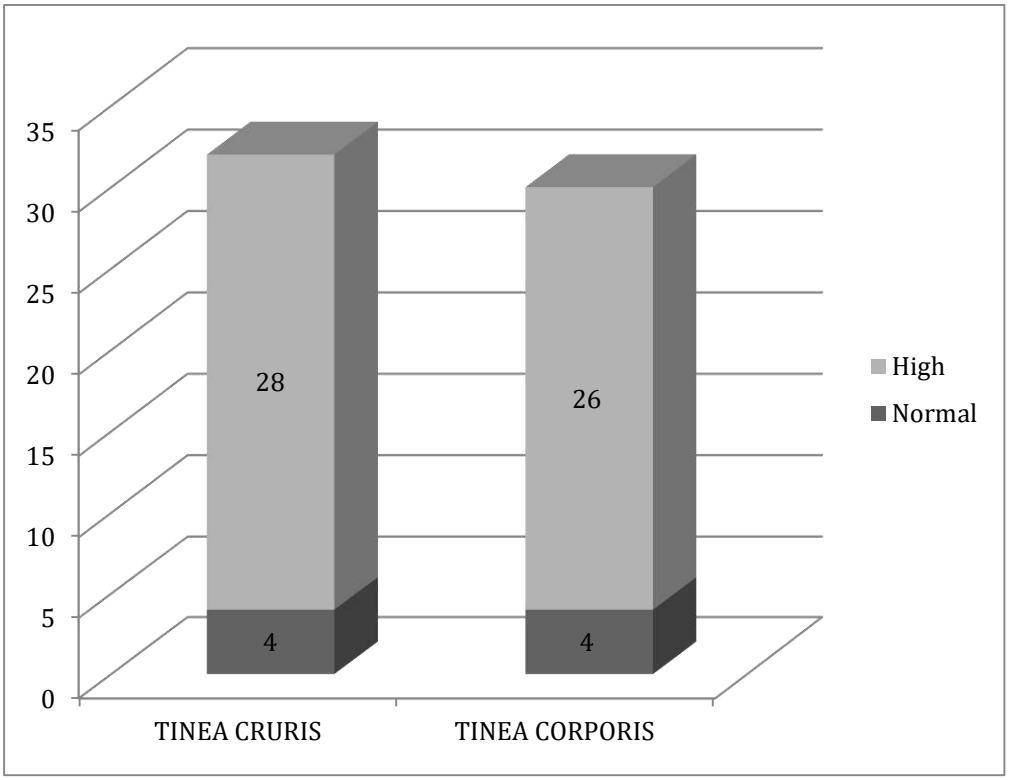

FIGURE 33: DISTRIBUTION OF PATIENTS WITH TINEA CRURIS AND TINEA CORPORIS ACCORDING TO WC

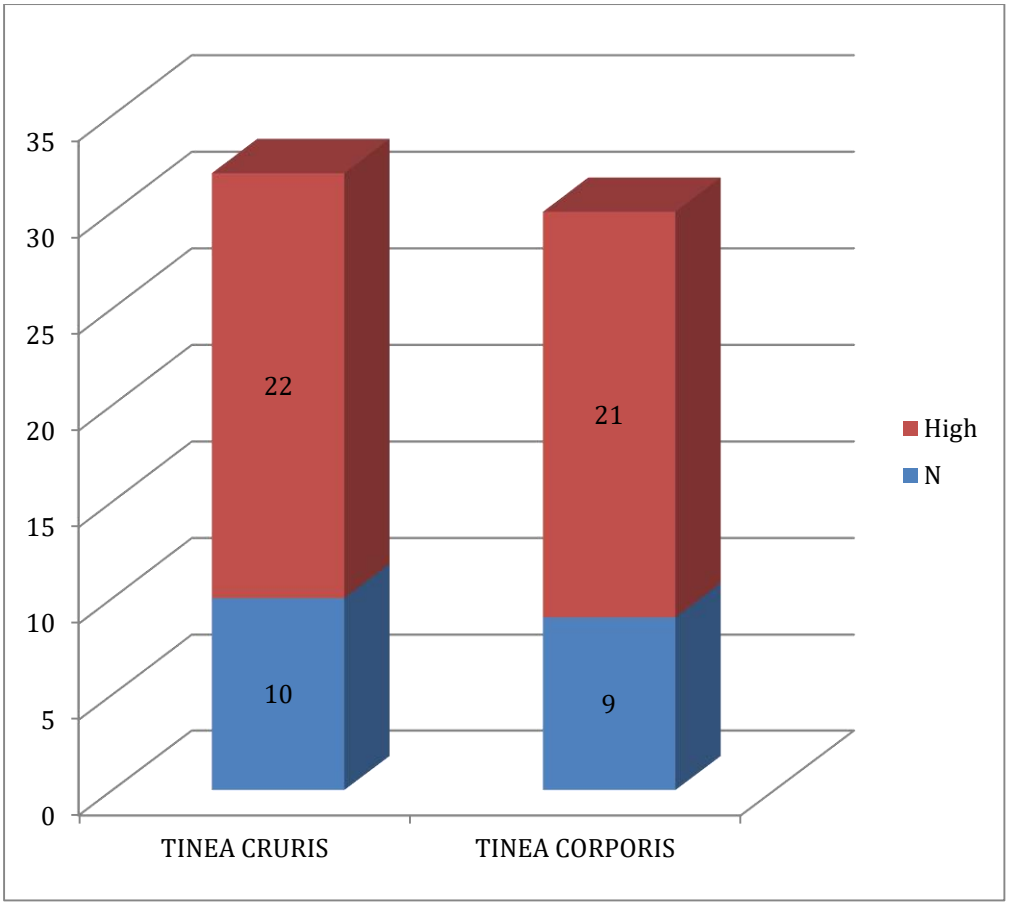

FIGURE 34: DISTRIBUTION OF PATIENTS WITH TINEA CRURIS AND TINEA CORPORIS ACCORDING TO WHR

TABLE 33: FREQUENCY OF FBS AND LIPID PROFILE ALTERATIONS IN PATIENTS WITH DERMATOPHYTIC INFECTIONS

	TC<200	TC>200	TG<200	TG>200	FBS<110	FBS>110
TINEA CRURIS	21	11	22	10	23	9
TINEA CORPORIS	18	12	22	8	25	5

ACANTHOSIS NIGRICANS

- In our study there were 24 cases of acanthosis nigricans, with male preponderance (79.17%). 18 out of 24 cases had grade II obesity and 95.80% cases had increased waist circumference.
- A significant proportion had impaired FBS and lipid profile as shown in table.
- Multiple site involvement was seen in patients. Neck(23) was most commonly involved followed by axilla(7) and face (1).

TABLE 34: GENDER DISTRIBUTION OF PATIENTS WITH ACANTHOSIS NIGRICANS

	Male(n)	Male(%)	Female(n)	Female(%)
Acanthosis Nigricans	19	79.17	5	20.83

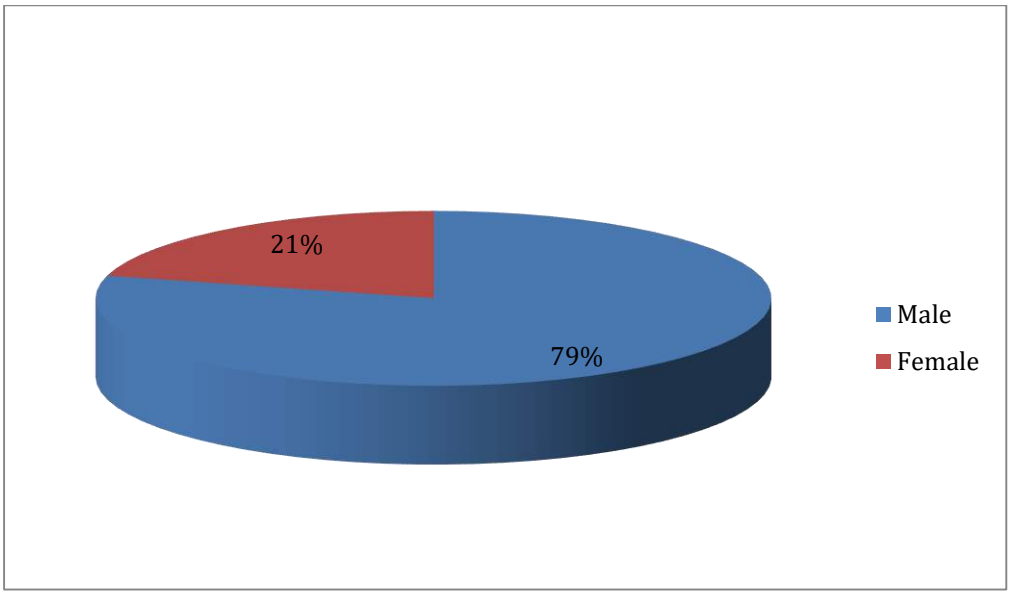

FIGURE 35: GENDER DISTRIBUTION OF PATIENTS WITH ACANTHOSIS NIGRICANS

TABLE 35: DISTRIBUTION OF PATIENTS WITH ACANTHOSIS NIGRICANS ACCORDING TO BMI, WC AND WHR

Dermatoses	Waist circumference		Waist-Hip Ratio		BMI	
	Normal	High	Normal	High	Grade I	Grade II
AN	1	23	9	15	6	18

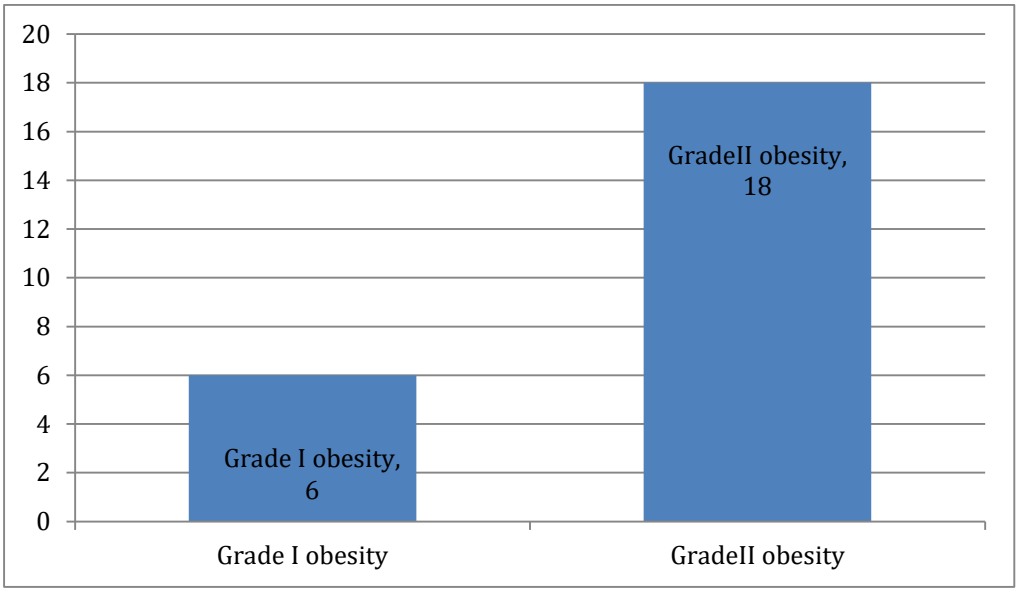

FIGURE 36: DISTRIBUTION OF PATIENTS WITH ACANTHOSIS NIGRICANS ACCORDING TO BMI

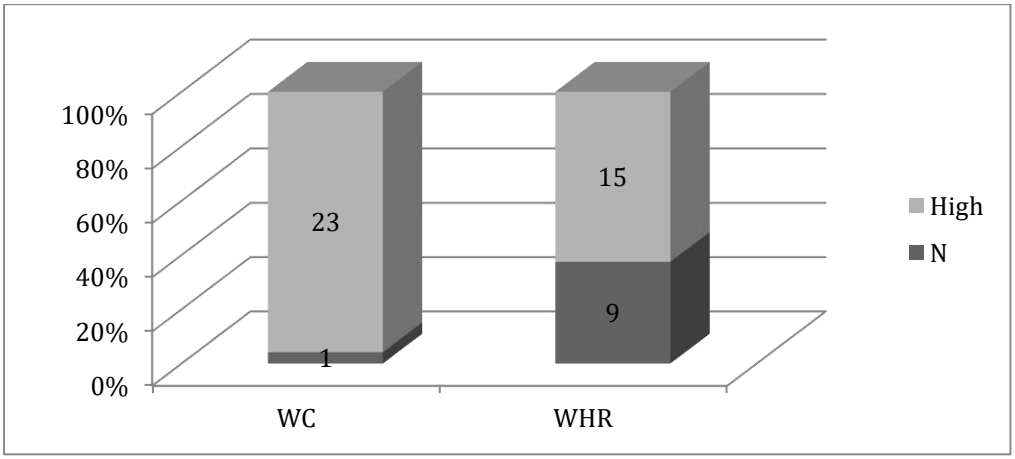

FIGURE 37: DISTRIBUTION OF PATIENTS WITH ACANTHOSIS NIGRICANS ACCORDING TO WC AND WHR

TABLE 36: FREQUENCY OF FBS AND LIPID PROFILE ALTERATIONS IN PATIENTS WITH ACANTHOSIS NIGRICANS

	TC<200	TC>200	TG<200	TG>200	FBS<110	FBS>110
AN	14	10	12	12	18	6

STRIAE DISTENSAE

- A total of 16 cases of striae were present in current study with 56.25% females.
- Waist circumference and WHR were increased in 100% cases with 15 out of 16 cases in grade II obesity patients.
- Various lab abnormalities are shown in table 39.

TABLE 37: GENDER DISTRIBUTION OF PATIENTS WITH STRIAE DISTENSAE

	Male(n)	Male(%)	Female(n)	Female(%)
STRIAE DISTENSAE	7	43.75	9	56.25

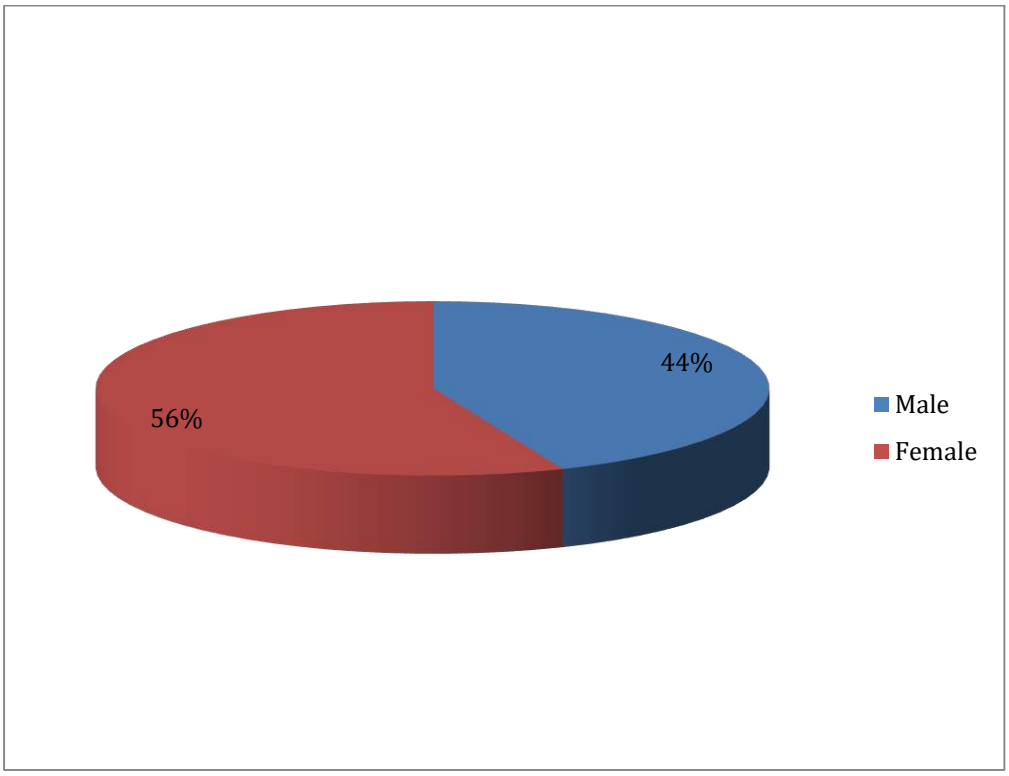

FIGURE 38: GENDER DISTRIBUTION OF PATIENTS WITH STRIAE DISTENSAE

TABLE 38: DISTRIBUTION OF PATIENTS WITH STRIAE DISTENSAE ACCORDING TO BMI, WC AND WHR

Dermatoses	Waist circumference		Waist-Hip Ratio		BMI	
	Normal	High	Normal	High	Grade I	Grade II
STRIAE DISTENSAE	0	16	0	16	1	15

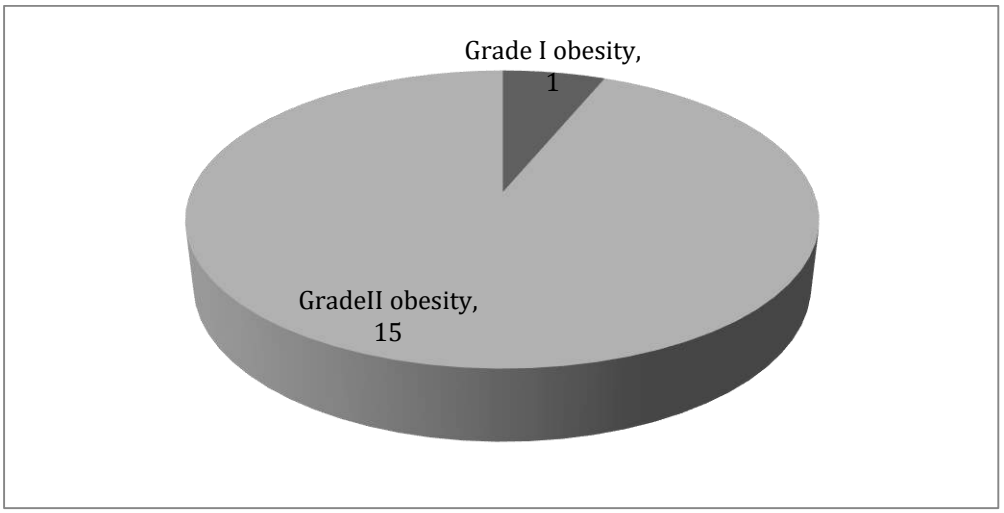

FIGURE 39: DISTRIBUTION OF PATIENTS WITH STRIAE DISTENSAE ACCORDING TO BMI

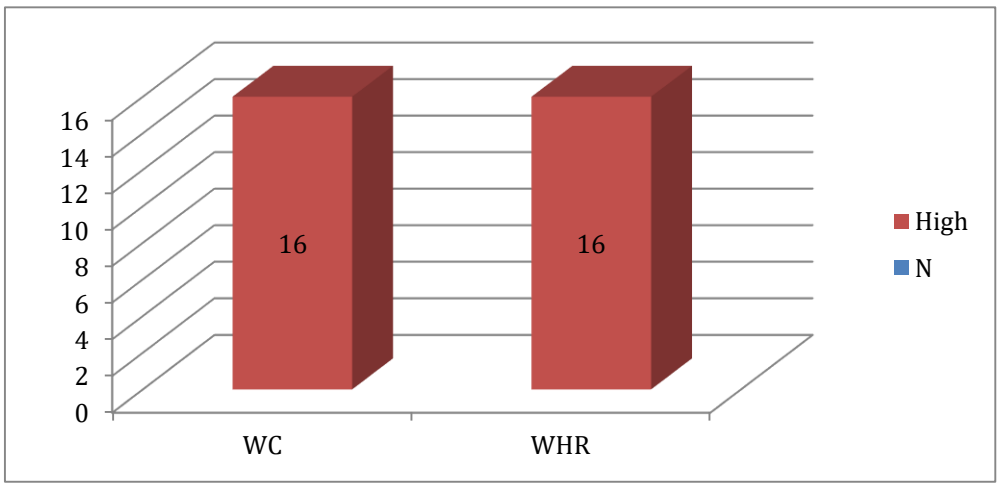

FIGURE 40: DISTRIBUTION OF PATIENTS WITH STRIAE DISTENSAE ACCORDING TO WC AND WHR

TABLE 39: FREQUENCY OF FBS AND LIPID PROFILE ALTERATIONS IN PATIENTS WITH STRIAE DISTENSAE

	TC<200	TC>200	TG<200	TG>200	FBS<110	FBS>110
STRIAE DISTENSAE	6	10	7	9	10	6

DISCUSSION

Obesity is an important healthcare problem in various industrialized and developing countries. It is characterised by an excess deposition of adipose tissue and is associated with a number of morbidities and mortality. Obesity can cause abnormalities in various functions of the skin, including sebum production, skin barrier function and sweat production, which can lead to a number of skin disorders in obese individuals.

In this study of 250 patients with obesity, the pattern and frequency of various cutaneous manifestations and their co-relation with anthropometric measurements (BMI, Waist circumference, waist-hip ratio), fasting lipid profile and fasting blood glucose levels were evaluated and compared.

AGE:

In our study, age group varies from 18 years to 81 years, with maximum number of patients in the age group of 41-50 years (27.60%) with mean age of 44.62 ±13.36 years which was comparable with the studies carried out by Boza et al[196] in which the mean age was 49.66 ± 15.61 years.

TABLE 40: COMPARISON OF MEAN AGE AMONG DIFFERENT STUDIES

	Khalil et al[197] (2011)	Boza et al[196] (2011)	Gomez et al[119] (2014)	Present study
Age (mean)	41.23±12.09 years (Males) 29.11±9.56 years (Females)	49.66 ± 15.61 years	38 ±14 years	44.62 ±13.36 years

GENDER:

In our study, there was slight female preponderance with129 (51.60%) females and 121 (48.40%) males. This female preponderance was also

seen in various studies done by Garcia et al[15], Boza et al[196] and Gomez et al[119] whereas male predominance was seen in a study done by Khalil et al[197].

TABLE 41: COMPARISON OF GENDER DISTRIBUTION AMONG DIFFERENT STUDIES

Gender distribution	Garcia et al[15] (1999)	Khalil et al[197] (2011)	Boza et al[196] (2011)	Gomez et al[119] (2014)	Present study
Females	126	32	56	91	129
Males	30	61	20	18	121

BMI:

In this study, a total of 67.20%(168) patients had grade I obesity and 32.80% (82) patients had grade II obesity. Mean BMI in our study was 29.27 ± 4.69 kg/m^2 as compared to 39.6 ± 8 kg/m^2 in a study by Gomez et al[119] and 39.37 ± 7.86 kg/m^2 in a study by Boza et al. [196]This difference in mean BMI was due to induction of patients with BMI 25.0-29.9 kg/m^2 in grade I obesity in our study, which was according to Asian guidelines for classification of obesity. A study done by Al-Mutairi[118] had 191 cases (45.80%) with BMI 25-29.9 kg/m^2 and 226 patients (54.20%) with BMI >30 kg/m^2.

In our study, acanthosis nigricans and striae distensae were strongly co-related with grade II obesity. Acanthosis nigricans was found to be a consistent finding in various other studies done in obese persons[198]Striae distensae was also a consistent finding in obese individuals which can be due to excessive tension on the skin caused by excessive weight.[199]

TABLE 42: COMPARISON OF MEAN BMI AMONG DIFFERENT STUDIES

	Khalil et al (2011)[197]	Boza et al[196] (2011)	Gomez et al[119] (2014)	Present study
Mean BMI (kg/m^2)	31.9±5.41 -males 31.23±4.97-females	39.37 ± 7.86 kg/m^2	39.6 ± 8 kg/m^2	29.27 ± 4.69 kg/m^2

WAIST CIRCUMFERENCE AND WAIST-HIP RATIO:

In our study, mean waist circumference was 97.73± 9.19 cm in contrast to 117±19 cm in study by Gomez et al. [119] Mean WHR was also measured in our study which was 0.95 ±0.05.

In our study, the association of higher waist circumference with acrochordons, pyoderma was statistically significant with p value of 0.019 and 0.030 respectively.

Similarly, in this study a higher WHR was associated with XP, striae distensae and pyoderma which was statistically significant with p-value of 0.011, 0.015 and 0.002 respectively.

TABLE 43: COMPARISON OF MEAN WHR AMONG DIFFERENT STUDIES

	Khalil et al[197]	Present study
Mean Waist:Hip ratio	0.95±0.09- males 0.90 ±0.05- females	0.95±0.05

DISEASE FREQUENCY:

In our study, acrochordons (24.58%) was the most common dermatoses which was comparable to the findings in the study carried out by Divyashree et al[200] but in contrast to findings in various other studies given in table 44. Other common cutaneous manifestations found in our study included xanthelasma palpebrum with 40 (11.30%), folliculitis with 35 (9.89%), intertrigo (9.60%), tinea cruris (9.04%), tinea

corporis (8.47%), acanthosis nigricans (6.78) and other manifestations in small proportion.

TABLE 44: COMPARISON OF MOST COMMON DERMATOSES SEEN IN DIFFERENT STUDIES

	Boza et al[196] (2011)	Khalil et al[197] (2011)	Gomez et al[119] (2014)	Divyashree et al[200] (2014)	Nisha K et al[201] (2017)	Present study
Most common dermatoses	Striae distensae > Acrochordons	Striae distensae> AN	AN > Acrochordons	Acrochordons> AN	AN > Striae distensae	Acrochordons > XP

LIPID PROFILE AND BLOOD GLUCOSE CHANGES:

In our study, 74 (29.60%) patients had dyslipidemia and an increased total cholesterol along with an abnormal raised levels of triglyceride and LDL in 65(26%) and 64 (25.60%) patients respectively which was in contrast to the study carried out by Gomez et al[119] in which hypercholesterolemia and hypertriglyceridemia was seen in 32% and 37% patients respectively. In a study conducted byBoza et al[196]**,** dyslipidemia was present in 27.6% of obese individuals which was similar to the findings in our study.

A raised total cholesterol was associated with striae distense and pyoderma which was statistically significant with p-value of 0.025 and 0.0001 respectively. An association of raised levels of triglycerides with striae distensae, acrochordons and folliculitis was

statistically significant with p-value of 0.041, 0.048 and 0.004 respectively. An association of hypertriglyceridemia and BMI with acrochordons has been reported in literature.[202,203]

The relation between obesity and skin infections has been well recognized in previous studies.[33,118] Various previous studies has found an increased risk of skin infections, mainly in the form of folliculitis, tinea cruris, tinea corporis and other cutaneous infections among obese individuals.

Fasting blood glucose in this study was deranged in 53 patients(21.20%), which was similar to findings in a study done by Boza et al[196] in which 23.7% patients of obesity had diabetes. Mean FBS was 101.38 ± 21.22 mg/dl. This was in contrast to the study conducted by Gomez et al[119] in which mean FBS was 95 ± 10 mg/dl. A statistically significant co-relation of raised FBS was seen with intertrigo (p-value=0.026). This can be attributed to increased risk of candidal infections in diabetics due to an immunosuppressive effect in these individuals.

TABLE 45: COMPARISON OF METABOLIC CHANGES AMONG DIFFERENT STUDIES

	Boza et al[196]	Present study
Dyslipidemia	27.6%	29.6%
Diabetes	23.7%	21.2%

SUMMARY

The present study was conducted to study various dermatological manifestations of obesity. A total of 250 obese patients were evaluated and detailed dermatological history and examination was carried out. The observations and results were tabulated and represented graphically. The summary of observations is as follows:

- Majority of patients were in 41-50 years age group (27.60%). Mean age was 44.62 ±13.36 years.
- A slight female preponderance was seen with 51.60% females compared to 48.40% males.
- A total of 67.20% patients were of grade I obesity and 32.80% patients of grade II obesity.
- Waist circumference was raised in 90% patients.
- Waist-Hip ratio was higher in 71.60% patients.
- Dyslipidemia was present in 29.60% patients and FBS was raised in 21.20% patients.
- Acrochordons(24.58%) was most common dermatoses in our study followed by xanthelasma palpebrum(11.30%), folliculitis(9.89%), intertrigo(9.60%).
- Other dermatoses like tinea, acanthosis nigricans, pyoderma, striae distensae, balanoposthitis, corns,onychomycosis, psoriasis vulgaris, cellulitis, oral thrush, chronic paronychia were also seen.
- Acrochordons was most common finding in our study, with slight female preponderance (52.87%). Neck was most common site to be affected followed by axilla. Most common association was with acanthosis nigricans followed by striae distense and Xanthelasma palpebrum.
- Acanthosis nigricans and striae distensae were significantly associated with grade II obesity (p-value-0.001 and 0.0001 respectively).
- Pyoderma and folliculitis were significantly associated with grade I obesity with P- value of 0.001.

- A statistically significant waist circumference was seen in acrochordons (p-value 0.019) and pyoderma (p-value 0.030). WHR was significantly associated with XP, pyoderma and striae distensae (p-value: 0.011, 0.002, 0.015 respectively).
- A statistically significant association with FBS was seen in intertrigo (p-value 0.026) and TC and TG with striae distensae (p-value 0.025 and 0.041 respectively).

CONCLUSION

Obesity is a major healthcare problem with deleterious effects on nearly all the systems of the body. The prevalence of obesity varies with a number of factors like age, gender, race, sex etc. Various physiological changes like excess body folds, increased sweating, increased blood flow, increased transepidermal water loss and the pressure effect can lead to numerous pathological dermatoses. A number of these dermatoses can be consistently associated with obesity and can even be considered as markers for the same.

With the increase in incidence of obesity, there is an increase in number of these accompanying cutaneous conditions. An understanding of underlying systemic disorder, can help in better management and avoidance of any sequelae. Understanding of various changes like lipid profile, FBS and their disease associations can be of use when it comes to treatment of these dermatoses as treatment of the underlying pathology of lipid alterations and glucose levels can be useful in controlling the skin condition.

An early recognition and proper attention to these dermatoses and their complications is cornerstone of appropriate management. A multi-disciplinary approach involving physician and dermatologist is important for tackling physical distress, co-morbidities and psychological disturbances associated with self image and confidence. Any treatment used for these dermatological conditions must include measures to for lifestyle modification for weight control.

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Picture 1A: Acanthosis Nigricans on neck

Picture 1B: Acanthosis Nigricans on neck

Picture 2A: Acanthosis Nigricans in axilla with Acrochordons

Picture 2B: Acanthosis Nigricans on neck with Acrochordons

Picture 2C: Acanthosis Nigricans in axilla with Acrochordons

Picture 3: Acrochordons on neck

Picture 4A: Xanthelasma Palpebrum

Picture 4B: Xanthelasma Palpebrum

Picture 5A: Intertrigo

Picture 5B: Intertrigo

Picture 6A: Tinea Corporis

Picture 6B: Tinea Corporis

Picture 6C: Tinea Corposris

Picture 6D: Tinea Corposris

Picture 7A: Striae Distensae

Picture 7B: Striae Distensae

Picture 8: Pyoderma

KEY TO MASTER CHART

AN	-	Acanthosis nigricans
DM	-	Diabetes mellitus
F	-	Female
FBS	-	Fasting blood glucose
HDL-C	-	High-density lipoprotein cholesterol
HS	-	Hidradenitis suppurativa
LDL	-	Low density lipoprotein
M	-	Male
N	-	NO
TG	-	Triglyceride
VVC	-	Vulvo-vaginal candidiasis
XP	-	Xanthelasma palperum
Y	-	Yes

S No.	MRD	Name	Age (Years)	Gender	Chief Complaints												Duration (days)	Associated disease	Sites involved								
					itching	redness	swelling	maceration	pigmentation	striae	yellow raised	pus discharge	skin tags	scales	pain	others			Inguinal	Axilla	Face	Lower limb	Upper limb	scalp	Neck	Eye lids	Trunk
1	208200	S	48	M	N	N	N	N	Y	Y	N	N	Y	N	N	N	240d	DM II	N	N	N	N	N	N	Y	N	Y
2	5680	SN	52	M	Y	Y	N	N	N	N	N	N	N	N	N	N	120d	N	Y	N	N	N	N	N	N	N	N
3	6829	SK	49	F	N	N	N	N	N	N	Y	N	Y	N	N	N	150d	N	N	N	N	N	N	N	Y	Y	N
4	885689	CS	50	M	N	N	N	N	N	N	Y	N	N	N	N	N	75d	N	N	N	Y	N	N	N	N	N	N
5	227017	JG	34	F	N	N	N	N	N	N	Y	N	N	N	N	N	360d	N	N	N	N	N	N	N	N	Y	N
6	281561	A	27	F	N	N	Y	N	N	N	N	Y	N	N	Y	N	4 d	N	N	N	Y	N	N	N	N	N	N
7	901936	S	35	F	N	N	N	N	N	N	Y	N	N	N	N	N	180d	N	N	N	N	N	N	N	N	Y	N
8	221347	HK	43	F	N	N	N	N	N	N	N	N	Y	N	N	N	540d	N	N	N	N	N	N	N	Y	N	N
9	914516	HS	48	M	Y	Y	Y	N	N	N	N	Y	N	N	Y	N	7d	N	N	N	N	N	Y	N	N	N	N
10	909377	A	39	F	N	N	N	N	N	N	Y	N	N	N	N	N	90d	N	N	N	N	N	N	N	N	Y	N
11	469728	MS	32	M	N	N	N	N	N	N	Y	N	N	N	N	N	60d	N	N	N	N	N	N	N	N	Y	N
12	927067	SS	46	M	Y	Y	N	Y	N	N	N	N	N	N	Y	N	60d	DM II	Y	N	N	N	N	N	N	N	N
13	194354	MS	66	M	Y	Y	N	N	Y	N	N	N	N	N	N	N	20d	N	Y	N	N	N	N	N	N	N	N
14	616387	BS	28	M	N	N	Y	N	N	N	N	Y	N	N	Y	N	10d	N	N	N	N	N	N	N	N	N	Y
15	811209	KK	38	F	N	N	N	N	N	N	Y	N	N	N	N	N	210d	N	N	N	N	N	N	N	N	Y	N
16	5947	SK	52	F	N	N	N	N	N	N	N	N	Y	N	N	N	240d	N	N	N	N	N	N	N	Y	N	N
17	901645	GK	24	F	N	Y	Y	N	N	N	N	Y	N	N	Y	N	7d	N	N	N	N	Y	N	N	N	N	N
18	437467	MS	29	M	Y	Y	N	N	N	N	N	N	Y	N	N	N	21d	N	N	N	Y	N	N	Y	Y	N	N
19	833762	DS	60	M	N	N	N	N	N	N	N	N	Y	N	N	N	720d	N	N	N	N	N	N	N	Y	N	N
20	182625	AK	36	F	N	N	N	N	N	N	Y	N	N	N	N	N	90d	N	N	N	N	N	N	N	N	Y	N
21	928887	D	32	M	Y	N	N	Y	N	N	N	N	N	N	Y	N	15d	N	Y	N	N	N	N	N	N	N	N
22	919377	JL	52	M	N	Y	N	N	N	N	N	N	Y	N	Y	N	5d	DM II	N	Y	N	N	N	N	Y	N	N
23	934719	HS	18	M	Y	Y	N	N	N	N	N	N	N	N	N	N	30d	N	Y	N	N	N	N	N	N	N	N
24	4693	KA	45	F	Y	Y	N	N	N	N	N	Y	N	N	Y	N	21d	N	N	N	N	N	N	N	N	N	Y
25	4235	NK	58	F	N	Y	Y	N	N	N	N	N	N	N	Y	N	3d	N	N	N	N	Y	N	N	N	N	N
26	219409	PK	58	F	N	N	N	Y	N	N	N	N	N	N	Y	N	20d	N	N	N	N	N	N	N	N	N	N
27	929828	BK	67	F	N	N	N	N	N	N	Y	N	Y	N	N	N	120d	N	N	N	N	N	N	N	Y	Y	N
28	928166	VK	36	M	Y	N	N	N	N	N	N	N	Y	N	N	N	30d	DM II	N	N	N	N	N	N	Y	N	N
29	73647	SS	48	M	Y	Y	N	N	N	N	N	N	Y	N	N	N	45d	N	Y	N	N	N	N	N	Y	N	N
30	43605	SS	38	M	Y	Y	N	N	N	N	N	N	N	N	N	N	60d	DM II	Y	N	N	N	N	N	Y	N	N
31	931697	SK	21	F	Y	Y	N	N	N	N	N	N	N	N	N	N	60d	N	Y	N	N	N	N	N	N	N	N
32	937576	HK	40	F	N	N	N	N	N	N	Y	N	N	N	N	N	210d	N	N	N	N	N	N	N	N	Y	N
33	7424	KK	49	F	N	N	Y	N	N	N	N	Y	N	N	Y	N	3d	N	N	N	N	N	N	N	N	N	Y
34	783937	KS	73	M	N	N	Y	N	N	N	N	Y	N	N	Y	N	4d	DM II	N	N	N	Y	N	N	N	N	N
35	900480	JS	44	M	N	N	Y	N	N	N	N	Y	N	N	Y	N	3d	N	N	Y	N	N	N	N	N	N	Y
36	947871	GK	31	F	Y	Y	N	Y	N	N	N	N	N	N	N	N	20d	N	N	N	N	N	N	N	N	N	N
37	944295	SP	40	M	Y	Y	N	N	N	N	N	N	N	N	N	N	90d	N	N	N	N	N	N	N	N	N	Y
38	948524	PK	52	F	Y	Y	N	N	N	N	N	N	N	N	N	N	150d	N	Y	N	N	N	N	N	N	N	Y
39	946256	SK	38	F	N	N	N	Y	N	N	N	N	Y	N	Y	N	60d	N	N	N	N	N	N	N	Y	N	N
40	946121	BS	56	M	N	N	N	N	Y	N	N	N	Y	N	N	N	364d	N	N	N	N	N	N	N	Y	N	N
41	947501	US	26	M	N	Y	Y	N	N	N	N	Y	N	N	Y	N	8d	N	N	N	N	Y	N	N	N	N	N
42	940771	CS	33	M	N	N	N	N	Y	N	N	N	N	N	N	ORAL ULCER	21d	N	N	Y	N	N	N	N	Y	N	N
43	946854	GS	40	M	Y	Y	N	N	N	N	N	N	Y	N	N	N	120d	N	Y	Y	N	N	N	N	Y	N	Y
44	43605	SS	38	M	Y	Y	N	N	Y	Y	N	N	Y	N	N	N	60d	DM II	Y	N	N	N	N	N	Y	N	Y
45	208204	SB	41	F	Y	Y	N	Y	N	N	N	Y	N	N	Y	N	7d	DM II	Y	N	N	N	N	N	N	N	N
46	955745	HK	40	F	N	N	Y	N	N	N	N	Y	N	N	Y	N	5d	N	N	Y	N	N	N	N	N	N	N
47	8136	AS	36	M	N	N	N	N	N	Y	N	N	Y	N	N	N	300d	N	N	N	N	N	N	N	Y	N	Y
48	4703	RKS	45	M	Y	N	N	Y	N	Y	N	N	N	N	Y	N	30d	N	Y	N	N	N	N	N	N	N	N
49	897400	AS	31	M	Y	Y	N	N	N	N	N	N	N	N	N	N	10d	N	Y	N	N	N	N	N	N	N	Y
50	950630	SK	60	F	N	N	N	N	N	N	Y	N	N	N	N	N	720d	N	N	N	N	N	N	N	N	Y	N
51	298934	SS	35	M	N	N	N	Y	N	N	N	N	N	N	Y	N	15d	DM II	Y	N	N	N	N	N	N	N	N
52	953000	BK	47	F	Y	Y	N	N	N	N	N	N	Y	N	N	N	90d	N	Y	Y	N	N	N	N	Y	N	Y
53	953150	CP	60	M	Y	N	N	Y	N	N	N	N	Y	N	Y	N	60d	N	Y	N	N	N	N	N	Y	N	N
54	953133	RK	49	M	Y	Y	N	N	N	N	Y	N	N	N	N	N	30d	N	Y	N	N	N	N	N	N	Y	Y
55	878507	BS	56	M	Y	N	N	Y	N	N	N	Y	N	N	Y	N	5d	N	N	N	N	N	N	N	N	N	N

S No.	MRD	Inframammary	Buttocks	WEB SPACES	Any other	Diagnosis	Height(m)	weight(kg)	BMI(kg/m²)	waist circumference (cm)	waist-hip ratio	Fasting lipid profile				FBS(mg/dl)
												Total Cholesterol	TG	HDL	LDL	
1	208200	N	N	N	N	AN, ACROCHORDONS , STRIAE DISTENSAE	1.73	98	32.7	106	1.03	172	184	40	96	275
2	5680	N	N	N	N	TINEA CRURIS	1.68	77	27.3	92	0.97	149	111	45	95	95
3	6829	N	N	N	N	ACROCHORDONS, XP	1.6	88	34.4	102	0.91	168	175	32	73	107
4	885689	N	N	N	N	ACROCHORDONS, XP	1.8	92	28.4	96	0.94	259	293	63	153	98
5	227017	N	N	N	N	XP	1.62	74	28.2	96	0.9	180	172	39	88	93
6	281561	N	N	N	N	FOLLICULITIS	1.6	73	28.5	82	0.95	156	74	45	106	92
7	901936	N	N	N	N	XP	1.62	75	28.6	102	0.91	150	139	49	85	93
8	221347	N	N	N	N	ACROCHORDONS	1.6	76	29.7	92	0.93	104	134	33	67	98
9	914516	N	N	N	N	FOLLICULITIS	1.71	78	26.7	102	0.92	200	60	48	117	102
10	909377	N	N	N	N	XP	1.64	76	28.3	92	0.95	140	210	140	120	84
11	469728	N	N	N	N	XP	1.8	88	27.2	96	0.96	243	168	48	160	108
12	927067	N	N	N	N	INTERTRIGO	1.8	102	31.5	110	1.03	320	368	76	102	149
13	194354	N	Y	N	N	TINEA CRURIS ET CORPORIS	1.8	84	25.9	86	0.93	102	135	45	42	82
14	616387	N	N	N	N	FOLLICULITIS	1.71	85	29.1	96	0.94	212	121	48	150	96
15	811209	N	N	N	N	XP	1.63	79	29.7	106	0.96	180	254	42	106	106
16	5947	N	N	N	N	ACROCHORDONS	1.58	72	28.8	94	0.92	193	145	40	130	107
17	901645	N	N	N	N	PYODERMA	1.6	68	26.6	76	0.92	126	107	45	59	82
18	437467	N	N	N	N	SEBORRHEIC DERMATITIS, ACROCHORDONS	1.72	76	25.7	92	0.95	174	133	46	101	92
19	833762	N	N	N	N	ACROCHORDONS	1.62	72	27.4	106	1.03	146	138	32	56	86
20	182625	N	N	N	N	XP	1.64	72	26.8	96	0.94	118	157	30	68	92
21	928887	N	N	N	N	INTERTRIGO	1.8	82	25.3	102	0.96	149	109	44	83	96
22	919377	N	N	N	GENITALIA	BALANOPOSTHITIS, ACROCHORDONS	1.75	83	27.1	112	1.05	207	360	43	127	132
23	934719	N	N	N	N	TINEA CRURIS	1.7	76	26.3	86	0.97	131	85	67	47	82
24	4693	N	N	N	N	PYODERMA	1.58	65	26	96	0.9	193	95	63	127	80
25	4235	N	N	N	N	FOLLICULITIS	1.6	83	32.4	110	0.91	231	174	49	166	102
26	219409	N	Y	N	N	INTERTRIGO	1.62	78	29.7	102	0.92	152	153	59	73	94
27	929828	N	N	N	N	ACROCHORDONS, XP	1.6	78	30.5	102	0.91	296	249	49	210	107
28	928166	N	N	N	N	GENERALISED PRURITUS, ACROCHORDONS	1.77	79	25.2	98	0.98	167	251	32	91	108
29	73647	N	N	N	N	TINEA CRURIS, ACROCHORDONS	1.82	86	26	102	0.96	194	340	47	116	98
30	43605	N	N	N	N	TINEA CRURIS, ACROCHORDONS	1.78	102	32.2	112	1.03	317	542	47	124	148
31	931697	N	N	N	N	TINEA CRURIS	1.64	74	27.5	82	0.91	147	110	42	80	84
32	937576	N	N	N	N	XP	1.67	78	28	86	0.93	180	154	36	83	97
33	7424	N	N	N	N	FOLLICULITIS	1.64	78	29	92	0.9	172	141	39	112	100
34	783937	N	N	N	N	FOLLICULITIS	1.77	83	26.5	96	0.94	194	156	57	84	148
35	900480	N	N	N	N	FOLLICULITIS	1.8	91	28.1	91	1.03	185	200	42	117	97
36	947871	N	N	Y	N	INTERTRIGO	1.65	68	25	92	0.92	203	174	45	78	82
37	944295	N	N	N	N	TINEA CORPORIS	1.8	85	26.2	96	0.94	151	136	39	89	102
38	948524	N	N	N	N	TINEA CRURIS ET CORPORIS	1.6	86	33.6	112	0.96	305	319	73	185	112
39	946256	N	N	Y	N	INTERTRIGO, ACROCHORDONS	1.65	72	26.4	86	0.93	148	159	76	40	102
40	946121	N	N	N	TOE NAILS	ONYCHOMYCOSIS, ACROCHORDONS	1.74	81	26.8	92	0.95	87	240	12	60	97
41	947501	N	N	N	N	PYODERMA	1.7	75	26	88	0.94	304	379	58	108	95
42	940771	N	N	N	TONGUE	ORAL THRUSH, AN	1.72	78	26.4	91	0.96	186	208	18	121	104
43	946854	N	Y	N	N	TINEA CRURIS ET CORPORIS, ACROCHORDONS	1.76	93	30	102	0.98	200	193	30	139	109
44	43605	N	N	N	N	TINEA CRURIS, AN, ACROCHORDONS, STRIAE DISTENSAE	1.8	107	33	122	1.05	317	542	47	124	128
45	208204	N	N	N	N	INTERTRIGO	1.57	68	27.6	102	0.91	190	371	37	104	108
46	955745	N	N	N	N	FOLLICULITIS	1.65	88	32.3	92	0.93	138	123	38	84	110
47	8136	N	N	N	N	ACROCHORDONS, STRIAE DISTENSAE	1.74	87	28.7	96	1.02	194	130	44	120	102
48	4703	N	N	N	N	INTERTRIGO, STRIAE DISTENSAE	1.76	95	90.7	96	1.04	207	400	34	122	98
49	897400	N	N	N	N	TINEA CRURIS ET CORPORIS	1.76	84	27.1	88	0.97	158	166	46	101	92
50	950630	N	N	N	N	XP	1.65	82	30.1	92	0.9	235	208	40	158	112
51	298934	N	N	N	N	INTERTRIGO	1.72	83	28.1	92	0.93	237	153	48	157	132
52	953000	N	N	N	N	TINEA CRURIS ET CORPORIS, ACROCHORDONS	1.62	91	34.1	112	0.96	169	169	30	111	112
53	953150	N	N	N	N	INTERTRIGO, ACROCHORDONS	1.78	86	27.1	96	0.94	153	114	56	81	110
54	953133	N	Y	N	N	TINEA CRURIS ET CORPORIS, XP	1.8	90	27.8	102	0.96	206	387	33	134	108
55	878507	N	N	N	SCROTUM	SCROTAL DERMATITIS	1.85	96	28	102	0.94	164	162	48	99	107

S No.	MRD	Name	Age (Years)	Gender	Chief Complaints												Duration (days)	Associated disease	Sites involved								
					itching	redness	swelling	maceration	pigmentation	striae	yellow raised	pus discharge	skin tags	scales	pain	others			Inguinal	Axilla	Face	Lower limb	Upper limb	scalp	Neck	Eye lids	Trunk
56	947826	R	20	F	N	N	N	N	N	N	N	N	Y	N	N	N	60d	N	N	N	N	N	N	N	Y	N	N
57	950391	MS	55	M	N	N	N	N	N	N	N	N	Y	N	N	N	300d	N	N	N	N	N	N	N	Y	N	N
58	3844	KK	48	F	N	N	N	N	Y	Y	Y	N	Y	N	N	N	120d	DM II	N	Y	N	N	N	N	Y	Y	Y
59	1119428	MK	25	F	N	N	N	N	N	N	Y	N	N	N	N	N	60d	N	N	N	N	N	N	N	N	Y	N
60	955183	AS	68	M	Y	Y	N	N	N	N	N	N	N	N	N	N	20d	N	N	N	N	N	N	N	N	N	Y
61	960845	SD	27	F	Y	Y	N	N	N	N	N	N	N	N	N	N	60d	N	Y	N	N	Y	Y	N	N	N	Y
62	920891	PK	45	F	Y	Y	N	N	N	N	Y	N	Y	N	N	N	92d	N	N	N	N	N	N	N	Y	Y	Y
63	957461	AS	61	M	Y	N	N	N	N	N	N	N	N	N	N	N	60d	N	Y	Y	N	Y	Y	Y	N	N	Y
64	957710	JK	52	F	Y	Y	N	N	N	N	N	N	N	N	N	N	30d	N	N	N	N	N	N	N	N	N	Y
65	193902	SD	75	F	Y	Y	N	Y	N	N	N	N	N	N	Y	N	14d	DM II	N	N	N	N	N	N	N	N	N
66	942747	H	43	F	Y	Y	Y	N	N	N	N	Y	N	N	Y	N	30d	N	Y	N	N	Y	N	N	N	N	Y
67	953711	GK	51	F	N	N	Y	N	N	N	Y	Y	N	N	Y	N	10d	DM II	N	N	N	N	N	N	N	Y	Y
68	951346	JK	58	F	Y	N	N	Y	N	N	N	N	Y	N	N	N	20d	DM II	Y	N	N	N	N	N	Y	N	N
69	505893	J	71	M	Y	Y	N	N	N	N	N	N	N	N	N	N	15d	N	Y	N	N	N	N	N	N	N	N
70	952381	C	55	F	N	N	Y	N	N	N	N	Y	Y	N	Y	N	6d	N	N	N	N	N	N	N	Y	N	Y
71	889531	R	46	F	N	Y	Y	N	N	N	N	Y	N	N	Y	N	7d	HTN	N	N	N	Y	N	N	N	N	N
72	955363	SS	38	M	N	Y	Y	N	Y	N	N	Y	Y	N	Y	N	3d	N	N	N	N	N	N	N	Y	N	Y
73	951916	GS	65	M	N	Y	Y	N	N	N	N	Y	N	N	Y	N	5d	DM II	N	N	N	N	N	Y	N	N	N
74	452553	RK	44	M	Y	Y	N	N	N	Y	N	N	Y	N	N	N	60d	DM II	Y	Y	N	N	N	N	Y	N	Y
75	952205	MD	43	M	N	N	N	N	N	N	N	N	N	N	Y	N	60d	N	N	N	N	N	N	N	N	N	N
76	952198	JR	62	M	Y	Y	N	N	N	N	N	N	N	N	Y	N	10d	DM II	N	N	N	N	N	N	N	N	N
77	940521	RK	50	M	Y	Y	N	N	N	N	N	Y	N	N	Y	N	3d	N	N	N	N	N	N	N	N	N	Y
78	65142	AK	37	F	Y	N	N	N	N	N	N	Y	N	N	N	N	5d	N	N	N	N	N	N	N	N	N	N
79	938660	AD	45	F	N	N	N	N	Y	Y	N	N	Y	N	N	N	180d	DM II	N	N	N	N	N	N	Y	N	Y
80	932683	MS	60	M	N	N	Y	N	N	N	N	Y	N	N	Y	N	10d	DM II	N	N	N	Y	N	N	N	N	N
81	955595	JS	40	M	Y	Y	N	N	N	N	N	N	N	N	Y	N	5d	DM II	N	N	N	N	N	N	N	N	N
82	220616	RK	46	F	N	N	N	N	N	N	N	N	Y	N	N	N	150d	N	N	N	N	N	N	N	Y	N	N
83	959650	HS	75	M	N	N	Y	N	Y	N	N	N	N	N	Y	N	45d	N	N	N	N	N	N	N	N	N	N
84	955336	BK	31	F	N	N	N	N	N	N	Y	N	N	N	N	N	60d	N	N	N	N	N	N	N	N	Y	N
85	35417	SK	52	M	Y	Y	N	N	N	N	N	N	N	N	N	N	20d	N	N	N	N	N	N	N	N	N	Y
86	4234	SK	53	F	N	Y	Y	N	N	N	N	Y	N	N	Y	N	7d	N	N	N	N	N	Y	N	N	N	N
87	273313	JK	67	F	N	N	N	N	N	N	Y	N	N	N	N	N	90d	N	N	N	N	N	N	N	N	Y	N
88	814774	HK	48	F	N	N	N	N	Y	Y	N	N	Y	N	N	N	90d	N	N	Y	N	N	N	N	Y	N	Y
89	961440	SK	41	M	N	N	N	N	Y	N	N	N	Y	N	N	N	60d	DM II	N	Y	N	N	N	N	Y	N	N
90	962496	MS	35	M	Y	Y	N	N	Y	N	N	N	Y	N	N	N	30d	DM II	Y	N	N	N	N	N	Y	N	Y
91	964603	DK	53	F	N	N	N	Y	N	N	N	N	Y	N	Y	N	42d	N	N	N	N	N	N	N	Y	N	N
92	3632	DK	42	F	N	N	N	N	N	N	Y	N	N	N	N	N	90d	N	N	N	N	N	N	N	N	Y	N
93	761942	MP	44	F	N	N	N	Y	N	N	Y	N	N	N	Y	N	25d	N	Y	N	N	N	N	N	N	Y	N
94	965809	N	40	F	N	Y	Y	N	N	Y	N	Y	Y	N	Y	N	10d	N	N	Y	N	N	N	N	Y	N	Y
95	960158	LPD	45	F	N	N	N	N	N	N	N	N	Y	N	N	N	60d	DM II	N	Y	N	N	N	N	Y	N	N
96	903505	SR	57	F	N	N	Y	N	N	N	N	Y	Y	N	Y	N	5d	DM II	N	N	N	N	N	N	Y	N	Y
97	914088	RR	70	F	N	N	N	Y	N	N	Y	N	Y	N	Y	N	12d	DM II	Y	N	N	N	N	N	Y	Y	N
98	381645	JK	42	F	N	N	Y	N	N	N	N	Y	N	N	Y	N	4d	N	N	N	N	Y	N	N	N	N	N
99	954712	LK	19	F	Y	N	N	N	N	N	N	N	N	N	N	GREASY SCALES	40d	N	N	N	N	N	N	Y	N	N	N
100	961853	SG	37	M	N	Y	Y	N	Y	N	N	Y	N	N	Y	N	7d	N	N	N	N	Y	N	N	Y	N	N
101	806867	GD	54	F	Y	Y	N	N	N	N	N	N	N	N	N	N	30d	N	N	N	N	Y	Y	N	N	N	Y
102	406500	HK	57	F	N	N	N	N	N	N	Y	N	Y	N	N	N	90d	DM II	N	N	N	N	N	N	Y	Y	N
103	225631	N	50	F	N	N	N	N	Y	Y	N	N	Y	N	N	N	150d	DM II	N	Y	N	N	N	N	Y	N	Y
104	800975	MK	50	F	N	N	N	N	N	Y	N	N	Y	N	N	N	120d	DM II	N	N	N	N	N	N	Y	N	Y
105	196255	SL	54	M	N	N	N	N	N	N	N	N	Y	N	N	N	90d	DM II	N	N	N	N	N	N	Y	N	N
106	973628	SS	40	F	N	N	N	Y	N	N	N	N	N	N	Y	N	20d	N	N	N	N	N	N	N	N	N	N
107	963735	NK	52	F	N	N	N	N	N	N	N	N	Y	N	N	N	45d	N	N	N	N	N	N	N	Y	N	N
108	973425	BS	66	M	N	N	Y	N	N	N	N	Y	N	N	Y	N	7d	N	N	N	N	Y	N	N	N	N	N
109	5168	BS	52	M	N	N	N	N	N	N	N	N	Y	N	N	N	90d	DM II	N	N	N	N	N	N	Y	N	N
110	980108	YM	20	M	N	N	N	N	Y	N	N	N	N	N	N	N	30d	N	N	N	Y	N	N	N	N	N	N

S No.	MRD	Inframammary	Buttocks	WEB SPACES	Any other	Diagnosis	Height(m)	weight(kg)	BMI(kg/m²)	waist circumference (cm)	waist-hip ratio	Fasting lipid profile				FBS(mg/dl)
												Total Cholesterol	TG	HDL	LDL	
56	947826	N	N	N	N	ACROCHORDONS	1.57	74	30	92	0.9	164	172	33	114	98
57	950391	N	N	N	N	ACROCHORDONS	1.78	82	25.9	96	0.94	173	133	27	119	92
58	3844	N	N	N	FINGER NAILS	ACRCOCHORDONS, XP, ONYCHOMYCOSIS, STRIAE DISTENSAE	1.61	80	30.9	106	0.94	217	204	40	140	132
59	1119428	N	N	N	N	XP	1.62	66	25.1	88	0.97	156	174	34	126	82
60	955183	N	N	N	N	TINEA CORPORIS	1.62	66	25.1	92	0.95	115	62	42	68	92
61	960845	N	Y	N	N	TINEA CRURIS ET CORPORIS	1.65	90	33.1	112	0.93	216	110	50	132	128
62	920891	N	Y	N	N	TINEA CORPORIS, XP, ACROCHORDONS	1.52	68	29.4	84	0.83	268	154	60	163	98
63	957461	N	Y	N	WHOLE BODY	GENERALISED PRURITUS	1.72	75	25.4	92	0.97	73	89	10	51	82
64	957710	N	N	N	N	TINEA CORPORIS	1.54	61	25.7	86	0.84	72	64	33	43	82
65	193902	N	Y	N	PERIANAL	INTERTRIGO	1.54	98	41.3	108	0.93	243	260	48	192	143
66	942747	N	Y	N	N	TINEA CRURIS ET CORPORIS, PYODERMA	1.62	75	28.6	96	0.86	212	251	51	138	95
67	953711	N	N	N	N	FOLLICULITIS, XP	1.61	68	26.2	99	0.89	149	203	20	85	106
68	951346	Y	N	N	N	INTERTRIGO, ACROCHORDONS	1.6	80	31.2	111	0.91	233	19	35	168	112
69	505893	N	N	N	N	TINEA CRURIS	1.77	90	28.7	108	1.05	128	146	38	82	101
70	952381	N	N	N	N	FOLLICULITIS, ACROCHORDONS	1.6	70	27.3	102	0.93	191	126	42	107	102
71	889531	N	N	N	N	FOLLICULITIS	1.6	76	29.7	106	0.96	187	162	48	107	98
72	955363	N	N	N	N	PYODERMA, AN, ACROCHORDONS	1.75	91	29.7	94	1.03	144	110	31	99	93
73	951916	N	N	N	N	SCALP FOLLICULITIS	1.77	88	28.1	92	0.98	175	172	35	82	118
74	452553	N	N	N	N	TINEA CRURIS, ACROCHORDONS, STRIAE DISTENSAE	1.79	96	30	98	1.03	148	154	42	75	122
75	952205	N	N	N	SOLES	CORNS	1.76	92	29.7	96	1.01	125	124	26	67	92
76	952198	N	N	N	GLANS	BALANOPOSTHITIS	1.75	88	28.7	95	0.94	154	115	33	101	142
77	940521	N	N	N	N	HERPES ZOSTER	1.72	75	25.4	86	0.97	133	124	46	66	93
78	65142	N	N	N	PER VAGINUM	VVC	1.61	73	27.8	86	0.93	143	96	49	90	88
79	938660	N	N	N	N	AN, ACROCHORDONS , STRIAE DISTENSAE	1.62	87	33.2	99	0.95	266	458	38	152	136
80	932683	N	N	N	N	PYODERMA	1.8	83	25.6	81	0.94	106	114	32	51	101
81	955595	N	N	N	GLANS	BALANOPOSTHITIS	1.8	96	29.6	102	1.04	190	200	47	103	112
82	220616	N	N	N	N	ACROCHORDONS	1.62	78	29.7	112	0.96	132	61	28	108	91
83	959650	N	N	N	TOE NAILS	ONYCHOMYCOSIS, CHRONIC PARONYCHIA	1.76	79	25.5	92	0.97	116	80	30	77	82
84	955336	N	N	N	N	XP	1.64	78	29	90	0.89	158	154	43	92	88
85	35417	N	N	N	N	TINEA CORPORIS	1.72	79	26.7	89	0.97	113	166	16	76	88
86	4234	N	N	N	N	FOLLICULITIS	1.52	72	31.2	96	0.96	140	163	25	83	112
87	273313	N	N	N	N	XP	1.6	78	30.5	102	0.91	202	241	37	115	92
88	814774	N	N	N	N	AN, ACROCHORDONS , STRIAE DISTENSAE	1.63	87	32.7	106	0.91	174	188	47	88	97
89	961440	N	N	N	N	ACROCHORDONS, AN	1.82	98	29.6	102	0.96	286	354	59	220	152
90	962496	N	N	N	N	TINEA CRURIS ET CORPORIS, AN, ACROCHORDONS	1.78	98	30.9	112	10.3	285	544	45	164	112
91	964603	N	N	Y	N	INTERTRIGO, ACROCHORDONS	1.64	88	32.7	112	0.96	216	403	39	131	122
92	3632	N	N	N	N	XP	1.58	71	28.4	82	0.81	202	89	47	122	88
93	761942	N	N	N	N	INTERTRIGO, XP	1.64	84	30.5	102	0.91	225	168	45	139	108
94	965809	N	N	N	N	FOLLICULITIS, STRIAE DISTENSAE, ACROCHORDONS	1.63	84	31.6	96	0.91	296	255	49	196	103
95	960158	N	N	N	TONGUE	CANDIDIASIS, ACROCHORDONS	1.59	81	32	92	0.93	223	161	39	149	117
96	903505	N	N	N	N	FOLLICULITIS, ACROCHORDONS	1.7	80	27.7	86	0.87	119	105	30	69	119
97	914088	Y	N	N	N	INTERTRIGO, XP, ACROCHODONS	1.62	79	30.1	90	0.91	180	186	56	99	122
98	381645	N	N	N	N	PYODERMA	1.65	78	28.7	86	0.93	164	91	47	93	88
99	954712	N	N	N	N	SEBORRHOEA CAPITIS	1.64	68	25.3	78	0.92	178	120	45	60	82
100	961853	N	N	N	N	PYODERMA, AN	1.76	87	28.1	96	0.95	141	166	38	113	104
101	806867	N	Y	N	N	TINEA CORPORIS	1.58	70	28	96	0.94	110	112	38	51	99
102	406500	N	N	N	N	ACROCHORDONS, XP	1.6	82	32	102	0.91	216	253	45	136	108
103	225631	N	N	N	N	AN, ACROCHORDONS, STRIAE DISTENSAE	1.57	75	30.4	106	0.94	240	340	38	170	108
104	800975	N	N	N	N	ACROCHORDONS, STRIAE DISTENSAE	1.6	79	30.9	106	0.92	180	197	42	147	213
105	196255	N	N	N	N	ACROCHORDONS	1.76	96	31	104	1.01	156	250	21	91	128
106	973628	N	N	Y	N	INTERTRIGO	1.57	68	27.6	96	0.94	172	147	36	54	82
107	963735	N	N	N	N	ACROCHORDONS	1.56	62	25.5	76	0.88	63	104	26	45	78
108	973425	N	N	N	N	PYODERMA	1.75	80	26.1	92	0.97	109	140	33	50	98
109	5168	N	N	N	N	ACROCHORDONS	1.72	88	29.7	102	0.92	129	180	46	110	128
110	980108	N	N	N	N	AN	1.82	112	33.8	102	1.06	198	350	35	128	98

S No.	MRD	Name	Age (Years)	Gender	Chief Complaints													Duration (days)	Associated disease	Sites involved								
					itching	redness	swelling	maceration	pigmentation	striae	yellow raised	pus discharge	skin tags	scales	pain	others				Inguinal	Axilla	Face	Lower limb	Upper limb	scalp	Neck	Eye lids	Trunk
111	368031	M	32	M	N	N	Y	N	N	N	N	Y	N	N	Y	N	5d	N	N	N	Y	N	N	N	N	N	N
112	375059	AK	32	F	N	N	N	N	N	N	N	Y	N	N	Y	N	4d	N	N	N	N	Y	N	N	N	N	N
113	852407	M	22	M	N	N	N	Y	N	N	N	N	N	N	Y	N	30d	N	N	N	N	N	N	N	N	N	N
114	972983	SS	62	M	Y	N	N	N	Y	N	N	N	Y	N	N	N	60d	N	N	N	N	N	N	N	Y	N	N
115	43605	SS	38	M	Y	Y	N	N	N	N	N	N	Y	N	N	N	25d	N	Y	N	N	N	N	N	Y	N	Y
116	980923	R	25	F	Y	Y	N	N	N	N	N	N	N	N	N	N	15d	N	N	N	N	N	N	N	N	N	Y
117	977188	AK	42	M	Y	Y	N	N	N	N	N	N	N	N	N	N	20d	N	Y	N	N	N	N	N	N	N	N
118	977576	PK	22	M	N	N	Y	N	N	N	N	Y	N	N	Y	N	5d	N	N	N	N	N	N	N	N	N	Y
119	971702	GK	45	F	N	N	Y	N	Y	N	N	N	N	N	Y	N	45d	N	N	N	N	N	N	N	N	N	N
120	976442	IS	31	M	N	N	N	N	N	N	Y	N	N	N	N	N	30d	N	N	N	N	N	N	N	N	Y	N
121	974011	GS	40	M	Y	Y	N	Y	N	N	N	N	N	Y	N	N	60d	N	Y	N	N	N	N	N	N	N	N
122	404082	A	32	M	Y	Y	N	N	N	N	N	N	Y	N	N	N	28d	N	Y	N	N	N	N	N	Y	N	Y
123	975106	RK	39	F	Y	Y	N	N	N	N	N	N	N	N	N	N	15d	N	N	N	N	N	N	N	N	N	N
124	554203	HS	52	M	N	N	N	N	Y	N	N	N	Y	N	N	N	180d	N	N	N	N	N	N	N	Y	N	N
125	980372	KB	38	F	Y	Y	N	N	Y	N	N	N	N	N	N	N	30d	N	N	N	N	N	N	N	N	N	N
126	913418	SS	30	M	N	N	N	N	N	N	Y	N	N	N	N	N	90d	N	N	N	N	N	N	N	N	Y	N
127	531770	MR	55	F	N	N	N	N	N	N	N	N	N	N	Y	ORAL ULCER	15d	N	N	N	N	N	N	N	N	N	N
128	975821	RS	40	M	Y	Y	N	Y	N	N	N	N	N	Y	Y	N	60d	N	Y	N	N	N	N	Y	N	N	N
129	982524	GS	31	M	Y	Y	N	N	Y	N	N	N	N	N	N	N	40d	N	Y	Y	N	N	N	N	Y	N	Y
130	206171	SK	22	F	N	N	Y	N	N	N	N	Y	N	N	Y	N	3d	N	N	N	N	N	Y	N	N	N	N
131	5818	PL	55	F	N	N	N	N	N	N	Y	N	N	N	N	N	120d	N	N	N	N	N	N	N	N	Y	N
132	993361	DC	62	M	Y	Y	N	N	Y	N	N	N	N	N	N	N	60d	N	N	N	N	Y	N	N	N	N	N
133	621262	NG	64	F	Y	Y	N	N	N	N	N	N	N	N	N	N	14d	N	Y	N	N	N	N	N	N	N	N
134	1004745	IS	32	M	Y	Y	N	N	N	N	N	N	N	Y	N	N	90d	N	N	N	N	N	Y	N	N	N	Y
135	367802	A	23	F	N	Y	Y	N	N	N	N	Y	N	N	Y	N	10d	N	N	N	N	Y	N	N	N	N	N
136	996811	G	48	F	Y	Y	N	N	N	N	N	N	N	N	N	N	14d	N	Y	N	N	N	N	N	N	N	N
137	1005574	PS	19	M	N	Y	N	Y	N	N	N	N	N	N	Y	N	2d	N	N	N	N	N	N	N	N	N	N
138	990116	PK	36	F	Y	Y	N	N	N	Y	N	N	N	N	N	N	21d	N	Y	N	N	N	N	N	N	N	Y
139	972697	SK	21	F	N	N	Y	N	N	N	N	Y	N	N	Y	N	3d	N	N	N	N	N	N	N	N	N	Y
140	994785	MD	40	M	N	Y	Y	N	N	N	N	Y	N	N	Y	N	5d	N	N	N	N	Y	N	N	N	N	N
141	276583	MK	31	F	N	N	Y	N	N	Y	N	Y	N	N	Y	N	4d	N	N	N	N	N	N	N	N	N	Y
142	5221	NK	42	M	N	N	N	N	N	N	N	N	Y	N	N	N	120d	N	N	N	N	N	N	N	Y	N	N
143	7061	BS	39	F	N	N	N	N	N	N	N	N	Y	N	N	N	60d	N	N	N	N	N	N	N	Y	N	N
144	190944	AC	57	F	N	Y	N	N	N	N	N	Y	N	N	Y	N	25d	DM II	N	N	N	Y	N	N	N	N	N
145	946717	IR	28	M	N	Y	Y	N	Y	Y	N	Y	Y	N	Y	N	20d	N	N	N	N	N	Y	N	Y	N	Y
146	387407	BS	61	M	N	N	N	N	N	N	N	N	Y	N	N	N	50d	N	N	N	N	N	N	N	Y	N	N
147	997498	SS	52	M	Y	Y	N	N	N	N	N	N	N	N	Y	N	180d	DM II	N	N	N	N	Y	N	N	N	Y
148	997288	NK	35	F	Y	Y	N	N	N	N	N	N	N	N	Y	N	14d	N	Y	N	N	N	N	N	N	N	N
149	990495	VD	55	F	Y	Y	N	N	N	N	N	N	N	N	Y	N	21d	N	N	N	N	N	N	N	N	N	Y
150	877784	PK	61	F	Y	Y	N	Y	N	N	N	N	N	N	Y	N	15d	N	Y	N	N	N	N	N	N	N	N
151	190639	B	47	M	N	Y	Y	N	N	N	N	Y	N	N	Y	N	10d	N	N	N	N	Y	N	N	N	N	N
152	1005160	VK	59	M	Y	Y	N	N	N	N	N	N	N	N	N	N	20d	DM II	Y	N	N	N	N	N	N	N	N
153	1001673	JS	41	F	Y	N	N	N	N	N	Y	N	N	N	N	WHITE PAPULES	45d	N	N	N	N	N	N	N	N	Y	Y
154	1007624	AB	35	F	N	N	N	N	N	N	Y	N	N	N	N	N	45d	N	N	N	N	N	N	N	N	Y	N
155	428900	L	46	F	N	Y	Y	Y	N	N	N	Y	N	N	Y	N	30d	N	N	N	N	N	N	Y	N	N	N
156	975694	N	26	F	Y	Y	N	N	N	N	N	N	N	Y	N	N	20d	N	N	N	N	N	N	N	N	N	N
157	194602	JS	58	M	N	N	Y	N	N	N	N	Y	N	N	Y	N	7d	N	N	N	N	Y	N	N	N	N	N
158	878840	AS	48	M	N	Y	N	Y	N	N	N	N	N	N	Y	N	20d	N	Y	N	N	N	N	N	N	N	N
159	375791	S	61	F	N	Y	N	Y	N	N	N	N	Y	N	Y	N	14d	N	N	N	N	N	N	N	Y	N	N
160	1013773	VD	72	F	N	Y	Y	N	N	N	N	Y	N	N	Y	N	15d	N	N	N	N	Y	N	N	N	N	N
161	1013087	PK	66	F	N	N	N	N	N	N	N	N	Y	N	N	N	120d	DM II	N	N	N	N	N	N	Y	N	N
162	226240	JK	44	F	N	N	N	N	N	N	Y	N	N	N	N	N	60d	N	N	N	N	N	N	N	N	Y	N
163	659871	HS	31	M	N	N	N	N	N	N	N	N	Y	N	N	N	30d	N	N	N	N	N	N	N	Y	N	N
164	47200	SS	34	M	N	Y	N	N	N	N	N	Y	N	N	Y	N	2d	N	N	N	N	N	N	Y	N	N	N
165	988548	MR	36	F	N	N	N	N	N	N	Y	N	N	N	N	N	30d	N	N	N	N	N	N	N	N	Y	N

S No.	MRD	Inframammary	Buttocks	WEB SPACES	Any other	Diagnosis	Height(m)	weight(kg)	BMI(kg/m²)	waist circumference (cm)	waist-hip ratio	Fasting lipid profile				FBS(mg/dl)
												Total Cholesterol	TG	HDL	LDL	
111	368031	N	N	N	N	FOLLICULITIS	1.78	85	26.8	92	0.95	221	236	48	148	88
112	375059	N	N	N	N	FOLLICULITIS	1.62	78	29.7	82	0.95	178	139	46	58	94
113	852407	N	N	Y	N	INTERTRIGO	1.72	77	26	92	0.97	226	297	40	161	88
114	972983	N	N	N	N	ACROCHORDONS, AN	1.76	96	31	106	1.08	151	257	20	94	96
115	43605	N	Y	N	N	TINEA CRURIS ET CORPORIS, ACROCHORDONS	1.75	91	29.7	104	1.01	317	542	47	124	112
116	980923	N	Y	N	N	TINEA CORPORIS	1.56	72	29.6	96	0.94	186	204	42	57	88
117	977188	N	N	N	N	TINEA CRURIS	1.72	94	31.8	90	0.93	104	111	30	70	82
118	977576	N	N	N	N	FOLLICULITIS	1.72	75	25.4	86	0.95	187	76	61	115	82
119	971702	N	N	N	FINGER NAILS	CHRONIC PARONYCHIA, ONYCHOMYCOSIS	1.65	93	34.2	96	0.93	148	95	44	85	85
120	976442	N	N	N	N	XP	1.77	85	27.1	96	1.01	134	203	34	76	92
121	974011	N	N	N	N	PSORIASIS VULGARIS	1.7	85	29.4	96	0.97	210	363	40	110	96
122	404082	N	Y	N	N	TINEACRURIS ET CORPORIS, ACROCHORDONS	1.62	78	29.7	102	1.04	324	376	55	202	98
123	975106	N	Y	N	N	TINEA CORPORIS	1.62	68	25.9	92	0.9	141	62	29	48	88
124	554203	N	N	N	N	ACROCHORDONS, AN	1.78	96	30.3	106	0.94	153	498	26	94	108
125	980372	N	Y	N	TOE NAILS	TINEA UNGUIM ET CORPORIS	1.54	62	26.1	86	1.04	102	85	23	67	83
126	913418	N	N	N	N	XP	1.8	86	26.5	102	1.02	226	133	54	141	88
127	531770	N	N	N	BUCCAL MUCOSA	ORAL THRUSH	1.52	68	29.4	106	0.94	157	214	42	72	97
128	975821	N	N	N	N	PSORIASIS VULGARIS	1.75	83	27.1	96	1.05	179	141	49	101	74
129	982524	N	N	N	N	TINEA CRURIS ET CORPORIS, AN	1.72	92	31.1	96	1.03	241	122	39	170	102
130	206171	N	N	N	N	FOLLICULITIS	1.62	72	27.4	81	0.94	115	128	32	73	82
131	5818	N	N	N	N	XP	1.57	76	30.8	102	0.94	181	297	42	111	110
132	993361	N	N	N	TOE NAILS	TINEA UNGUIM ET CORPORIS	1.74	85	28.1	98	1.02	270	164	56	151	98
133	621262	N	N	N	N	TINEA CRURIS	1.54	65	27.4	102	0.92	117	91	29	75	92
134	1004745	N	N	N	N	PSORIASIS VULGARIS	1.82	92	27.8	106	1.03	194	156	41	121	78
135	367802	N	N	N	N	ACUTE PARONYCHIA, CORNS	1.54	76	32	76	0.92	124	112	35	81	81
136	996811	N	N	N	N	TINEA CRURIS	1.6	68	26.6	92	0.95	137	178	24	89	102
137	1005574	N	N	N	GLANS	HERPES GENITALIS, BALANOPOSTHITIS	1.77	80	25.5	82	0.95	158	74	41	101	98
138	990116	N	N	N	N	TINEA CRURIS ET CORPORIS, STRIAE DISTENSAE	1.62	85	32.4	106	0.94	264	218	44	173	92
139	972697	N	N	N	N	FOLLICULITIS	1.62	6	25.1	76	0.9	154	59	48	81	78
140	994785	N	N	N	N	PYODERMA	1.75	82	26.8	96	0.97	201	149	45	115	106
141	276583	N	N	N	N	STRIAE DISTENSAE, FOLLICULITIS	1.52	74	32	92	0.95	212	97	57	125	87
142	5221	N	N	N	N	ACROCHORDONS	1.75	85	27.8	106	1.1	231	291	144	43	98
143	7061	N	N	N	N	ACROCHORDONS	1.6	74	28.9	96	0.94	141	139	77	41	95
144	190944	N	N	N	N	NON HEALING ULCER	1.65	88	32.3	102	0.96	186	162	107	47	110
145	946717	N	N	N	N	AN, ACROCHORDONS,STRIAE DISTENSAE, FOLLICULITIS	1.8	98	30.2	106	1.03	238	547	38	138	88
146	387407	N	N	N	N	ACROCHORDONS	1.75	84	27.4	92	0.95	174	96	42	103	88
147	997498	N	N	N	N	PSORIASIS VULGARIS	1.86	98	28.3	102	1.06	204	119	50	144	104
148	997288	N	N	N	N	TINEA CRURIS	1.62	78	29.7	102	0.96	87	158	28	39	98
149	990495	N	N	N	N	TINEA CORPORIS	1.52	77	33.3	101	0.95	206	152	30	80	88
150	877784	N	N	N	N	INTERTRIGO	1.54	66	27.8	96	0.92	117	122	32	60	86
151	190639	N	N	N	N	PYODERMA	1.75	83	27.1	86	0.97	107	61	29	49	98
152	1005160	N	N	N	N	TINEA CRURIS	1.72	84	28.4	96	1.04	110	129	31	68	112
153	1001673	N	N	N	N	XP, MOLLUSCUM CONTAGIOSUM	1.58	76	30.4	106	0.92	183	119	45	110	93
154	1007624	N	N	N	N	XP	1.6	78	30.5	86	0.93	175	113	50	112	98
155	428900	N	N	Y	N	INTERTRIGO, FOLLICULITIS	1.72	82	27.7	96	1.04	148	59	44	100	92
156	975694	N	Y	N	N	TINEA CORPORIS	1.58	65	26	72	0.87	132	48	82	49	78
157	194602	N	N	N	N	FOLLICULITIS	1.78	84	26.5	86	0.97	143	110	25	102	98
158	878840	N	N	N	N	INTERTRIGO	1.75	77	25.1	86	0.93	142	91	41	93	88
159	375791	N	Y	N	N	INTERTRIGO, ACROCHORDONS	1.6	74	28.9	96	0.9	204	127	53	133	98
160	1013773	N	N	N	N	PYODERMA	1.6	65	25.4	88	0.89	71	375	10	52	95
161	1013087	N	N	N	N	ACROCHORDONS	1.63	78	29.4	96	0.9	167	80	45	106	123
162	226240	N	N	N	N	XP	1.62	83	31.6	102	0.91	206	191	50	118	102
163	659871	N	N	N	N	ACROCHORDONS	1.72	85	28.7	96	1.04	284	263	54	177	106
164	47200	N	N	N	N	FOLLICULITIS	1.75	84	27.4	92	0.95	184	119	36	124	92
165	988548	N	N	N	N	XP	1.6	74	28.9	96	94	175	118	47	105	90

S No.	MRD	Name	Age (Years)	Gender	Chief Complaints												Duration (days)	Associated disease	Sites involved								
					itching	redness	swelling	maceration	pigmentation	striae	yellow raised	pus discharge	skin tags	scales	pain	others			Inguinal	Axilla	Face	Lower limb	Upper limb	scalp	Neck	Eye lids	Trunk
166	989893	SM	25	M	N	N	Y	N	N	N	N	Y	N	N	Y	N	20d	N	N	N	N	N	N	N	N	N	Y
167	995426	BK	54	F	N	N	N	N	N	N	Y	N	Y	N	N	N	90d	N	N	N	N	N	N	N	Y	Y	N
168	1017465	KK	55	F	N	N	N	N	N	N	Y	N	Y	N	N	N	60d	N	N	N	N	N	N	N	Y	Y	N
169	833762	DS	61	M	N	N	N	N	N	N	N	N	Y	N	N	N	120d	N	N	N	N	N	N	N	Y	N	N
170	988408	RK	35	F	N	N	N	N	N	Y	N	N	Y	N	N	N	30d	N	N	N	N	N	N	N	Y	N	Y
171	533805	SK	48	F	N	Y	N	N	N	N	N	Y	N	N	Y	N	6d	N	N	N	N	Y	N	N	N	N	N
172	1011603	PS	52	M	Y	Y	N	N	N	N	N	N	N	Y	N	N	20d	N	N	N	N	N	N	N	N	N	Y
173	1012511	M	23	M	N	Y	Y	N	N	N	N	Y	N	N	Y	N	10d	DM II	N	N	N	N	N	N	N	N	N
174	738616	AK	27	F	N	N	N	Y	N	N	N	N	N	N	Y	N	15d	N	Y	N	N	N	N	N	N	N	N
175	35417	SK	52	M	N	N	Y	N	N	N	N	Y	N	N	Y	N	4d	DM II	N	N	N	N	N	N	N	N	Y
176	3943	NK	49	F	N	N	N	N	N	N	N	N	Y	N	N	N	60d	N	N	N	N	N	N	N	Y	N	N
177	843438	N	19	M	N	N	N	N	Y	N	N	N	N	N	N	N	60d	N	N	N	N	N	N	N	Y	N	N
178	761790	RN	64	M	N	N	N	Y	N	N	N	N	N	N	Y	N	15d	DM II	N	N	N	N	N	N	N	N	N
179	1008698	SK	45	F	N	Y	Y	N	N	N	N	N	N	N	Y	N	7d	N	N	N	N	Y	N	N	N	N	N
180	1018060	KK	37	F	Y	Y	N	N	N	N	N	N	Y	Y	N	N	30d	N	N	N	N	N	Y	N	Y	N	Y
181	7637	PK	37	F	N	N	N	N	Y	N	N	N	Y	N	N	N	45d	N	N	N	N	N	N	N	Y	N	N
182	1013429	BS	42	M	N	N	N	N	N	N	N	N	Y	N	N	N	90d	DM II	N	N	N	N	N	N	Y	N	N
183	218669	JK	51	F	N	Y	Y	N	N	N	N	Y	N	N	Y	N	5d	N	N	N	N	N	Y	N	N	N	N
184	234938	DB	29	F	N	Y	Y	N	N	N	N	Y	N	N	Y	N	3d	N	N	N	N	Y	N	N	N	N	N
185	1022950	RS	58	F	N	N	N	N	N	N	N	N	Y	N	N	CALLOSITY	180d	DM II	N	N	N	N	N	N	Y	N	N
186	1023408	JK	50	F	N	N	N	N	N	N	Y	N	Y	N	N	N	120d	N	N	N	N	N	N	N	Y	Y	N
187	1024432	SS	32	M	Y	N	N	Y	N	N	N	N	Y	N	Y	N	20d	N	Y	Y	N	N	N	N	Y	N	N
188	49842	S	43	F	N	N	N	N	N	N	Y	N	N	N	N	N	60d	N	N	N	N	N	N	N	N	Y	N
189	996158	S	40	F	N	N	N	N	N	N	N	N	Y	N	N	N	75d	N	N	N	N	N	N	N	Y	N	N
190	675803	BS	46	M	Y	N	N	Y	N	N	N	N	N	N	Y	N	12d	DM II	Y	N	N	N	N	N	N	N	N
191	597352	BK	49	F	N	N	N	N	N	N	Y	N	Y	N	N	N	120d	N	N	N	N	N	N	N	Y	Y	N
192	974034	MG	36	F	N	N	N	N	N	N	Y	N	N	N	N	N	60d	N	N	N	N	N	N	N	N	Y	N
193	908073	DA	51	M	N	N	N	N	Y	N	N	N	N	N	N	N	90d	N	N	Y	N	N	N	N	Y	N	N
194	40585	AL	81	M	Y	Y	Y	N	N	N	N	N	N	N	Y	N	10d	DM II	N	N	N	Y	N	N	N	N	N
195	998762	KR	36	M	Y	Y	N	N	N	N	N	N	N	Y	N	N	14d	N	Y	N	N	N	N	N	N	N	N
196	314712	SS	18	M	Y	Y	N	N	N	N	N	N	N	Y	N	N	10d	N	N	N	N	N	N	N	N	N	Y
197	718060	SS	33	M	N	N	N	N	Y	N	N	N	Y	N	N	N	90d	N	N	N	N	N	N	N	Y	N	N
198	1015707	MKJ	23	F	N	Y	Y	N	N	N	N	Y	N	N	Y	N	5d	N	N	N	N	N	Y	N	N	N	N
199	1022945	AS	50	M	N	N	N	N	N	N	N	N	Y	N	N	N	150d	DM II	N	Y	N	N	N	N	Y	N	N
200	1023415	JP	36	M	N	N	N	N	Y	N	N	N	Y	N	N	N	60d	N	N	N	N	N	N	N	Y	N	N
201	1024603	MK	30	F	N	N	Y	N	N	N	N	N	N	N	Y	N	20d	N	N	N	N	N	N	N	N	N	N
202	641577	JS	19	M	N	N	Y	Y	N	N	N	Y	N	N	Y	N	30d	N	N	Y	N	N	N	N	N	N	N
203	1032222	RS	41	M	N	N	N	N	Y	N	N	N	Y	N	N	N	75d	N	N	N	N	N	N	N	Y	N	N
204	1019278	TJ	38	F	N	N	N	N	N	N	N	N	N	N	N	PAPULES	30d	N	N	N	Y	N	N	N	N	N	N
205	983901	GS	42	M	Y	Y	N	N	N	N	N	N	N	Y	N	N	25d	N	Y	N	N	N	N	N	N	N	Y
206	234113	HK	20	M	N	Y	Y	N	N	N	N	Y	N	N	Y	N	3d	N	N	N	N	N	N	Y	N	N	N
207	1033101	SS	48	F	N	N	N	N	N	N	Y	N	Y	N	N	N	30d	DM II	N	N	N	N	N	N	Y	Y	N
208	1022849	KS	42	M	N	N	N	Y	N	N	N	N	N	N	Y	N	20d	N	Y	N	N	N	N	N	N	N	N
209	723350	S	34	M	N	Y	Y	N	N	N	N	Y	N	N	Y	N	10d	N	N	N	N	Y	N	N	N	N	N
210	7534	DP	39	M	N	N	N	N	N	N	N	N	Y	N	N	N	45d	N	N	N	N	N	N	N	Y	N	N
211	1032393	SK	43	M	Y	Y	N	N	N	N	N	N	N	Y	N	N	14d	N	Y	N	N	N	N	N	N	N	N
212	1110661	DS	20	M	N	N	Y	N	N	N	N	Y	N	N	N	N	3d	N	N	N	N	N	N	N	N	N	Y
213	1051001	D	32	F	N	N	N	N	N	N	N	N	Y	N	N	N	30d	N	N	N	N	N	N	N	Y	N	N
214	134262	AK	64	F	Y	Y	N	N	N	N	N	N	N	Y	N	N	10d	N	Y	N	N	N	N	N	N	N	N
215	1054229	MM	40	M	N	N	N	N	N	N	Y	N	N	N	N	N	60d	N	N	N	N	N	N	N	N	Y	N
216	1028445	AS	69	M	Y	N	N	Y	N	N	N	N	N	N	Y	N	14d	DM II	Y	N	N	N	N	N	N	N	N
217	1054430	GK	45	F	Y	N	N	Y	N	N	N	N	N	N	Y	N	10d	N	N	N	N	N	N	N	N	N	N
218	1043866	KC	32	F	N	Y	Y	N	N	N	N	N	Y	N	Y	N	10d	N	N	N	N	N	N	N	Y	N	Y
219	1044710	KD	45	F	N	N	N	N	N	N	N	N	Y	N	N	N	120d	N	N	N	N	N	N	N	Y	N	N
220	1025863	KS	25	M	Y	Y	N	N	N	N	N	N	N	Y	N	N	20d	N	Y	N	N	N	N	N	N	N	N

S No.	MRD	Inframammary	Buttocks	WEB SPACES	Any other	Diagnosis	Height(m)	weight(kg)	BMI(kg/m²)	waist circumference (cm)	waist-hip ratio	Fasting lipid profile				FBS(mg/dl)
												Total Cholesterol	TG	HDL	LDL	
166	989893	N	N	N	N	FOLLICULITIS	1.72	86	29.1	96	1.02	202	148	47	125	98
167	995426	N	N	N	N	ACROCHORDONS, XP	1.62	86	32.8	106	0.94	217	289	44	131	104
168	1017465	N	N	N	N	ACROCHORDONS, XP	1.54	74	31.2	102	0.96	165	197	40	85	97
169	833762	N	N	N	N	ACROCHORDONS	1.76	86	27.8	102	0.104	209	285	45	123	112
170	988408	N	N	N	N	ACROCHORDONS, STRIAE DISTENSAE	1.6	86	33.6	116	1.03	240	205	50	181	98
171	533805	N	N	N	N	FOLLICULITIS	1.6	75	29.3	96	0.9	138	81	105	40	78
172	1011603	N	Y	N	N	TINEA CORPORIS	1.75	95	31	106	1.1	169	148	46	110	96
173	1012511	N	Y	N	N	PYODERMA	1.78	85	26.8	92	0.95	181	186	35	119	117
174	738616	N	N	N	N	INTERTRIGO	1.62	76	29	82	0.95	186	62	91	104	72
175	35417	N	N	N	N	FOLLICULITIS	1.7	81	28	96	1.04	154	151	27	97	134
176	3943	N	N	N	N	ACROCHORDONS	1.58	76	30.4	102	0.96	222	203	38	62	78
177	843438	N	N	N	N	AN	1.76	88	28.4	96	1.04	177	276	21	62	83
178	761790	N	Y	N	N	INTERTRIGO	1.76	80	25.8	92	1.04	168	94	34	123	136
179	1008698	N	N	N	N	CELLULITIS	1.6	74	28.9	96	0.9	127	155	24	77	81
180	1018060	N	N	N	N	TINEA CORPORIS, ACROCHORDONS	1.6	80	31.2	102	0.91	193	162	48	128	93
181	7637	N	N	N	N	AN , ACROCHORDONS	1.58	77	30.8	96	0.9	199	105	40	151	87
182	1013429	N	N	N	N	ACROCHORDONS	1.82	96	29	106	1.03	221	137	57	146	112
183	218669	N	N	N	N	FOLLICULITIS	1.6	74	28.9	96	0.9	163	136	44	100	95
184	234938	N	N	N	N	FOLLICULITIS	1.6	68	26.6	86	0.89	165	116	33	124	83
185	1022950	N	N	N	SOLES	CORNS, ACROCHORDONS	1.62	84	32	106	0.94	195	149	45	131	111
186	1023408	N	N	N	N	XP, ACROCHORDONS	1.6	78	30.5	96	0.94	169	154	49	110	109
187	1024432	N	N	N	N	INTERTRIGO, ACROCHORDONS	1.77	93	29.7	102	1.04	191	200	49	132	98
188	49842	N	N	N	N	XP	1.58	78	31.2	106	1.03	246	295	46	156	106
189	996158	N	N	N	N	ACROCHORDONS	1.6	78	30.5	92	0.95	139	183	33	73	104
190	675803	N	N	N	N	INTERTRIGO	1.75	92	30	112	1.05	149	168	39	75	107
191	597352	N	N	N	N	XP, ACROCHORDONS	1.62	84	32	106	0.94	212	282	56	174	87
192	974034	N	N	N	N	XP	1.62	78	29.7	92	0.95	181	186	34	121	98
193	908073	N	N	N	N	AN	1.75	96	31.3	112	1.09	184	144	37	130	97
194	40585	N	N	N	N	CELLULITIS, ASTEOTIC ECZEMA	1.75	78	25.5	86	1.04	137	121	44	69	122
195	998762	N	N	N	N	TINEA CRURIS	1.78	88	27.8	96	1.04	180	188	49	115	78
196	314712	N	N	N	N	TINEA CORPORIS	1.76	78	25.2	82	0.95	139	102	41	37	75
197	718060	N	N	N	N	ACROCHORDONS WITH AN	1.78	97	30.6	106	1.03	223	202	37	145	97
198	1015707	N	N	N	N	FOLLICULITIS	1.62	68	25.9	76	0.82	150	116	56	92	78
199	1022945	N	N	N	N	ACROCHORDONS	1.85	105	30.7	116	1.03	235	249	39	167	107
200	1023415	N	N	N	N	ACROCHORDONS, AN	1.8	103	31.8	112	0.94	193	191	97	139	109
201	1024603	N	N	N	SOLES	CORNS	1.65	77	28.3	82	0.85	204	116	61	140	81
202	641577	N	N	N	N	HS	1.75	79	25.8	82	0.89	168	155	41	104	73
203	1032222	N	N	N	N	ACROCHORDONS, AN	1.78	110	34.7	112	0.91	168	155	41	104	107
204	1019278	N	N	N	N	PLANE WARTS	1.58	76	30.4	106	0.91	230	374	58	121	109
205	983901	N	Y	N	N	TINEA CRURIS ET CORPORIS	1.8	96	29.6	116	1.03	142	112	40	96	97
206	234113	N	N	N	N	FOLLICULITIS	1.78	82	25.9	86	0.84	197	126	46	122	78
207	1033101	N	N	N	N	ACROCHORDONS, XP	1.62	82	31.2	112	0.92	246	280	42	148	151
208	1022849	N	N	N	N	INTERTRIGO	1.78	94	29.7	112	0.96	194	122	45	144	104
209	723350	N	N	N	N	PYODERMA	1.75	82	26.8	92	0.9	158	144	44	98	95
210	7534	N	N	N	N	ACROCHORDONS	1.7	84	29.1	102	0.96	176	81	40	128	102
211	1032393	N	N	N	N	TINEA CRURIS	1.68	74	26.2	96	0.94	143	138	27	99	98
212	1110661	N	N	N	N	FOLLICULITIS	1.78	88	27.8	92	0.86	154	186	38	76	82
213	1051001	N	N	N	N	ACROCHORDONS	1.62	76	29	92	0.86	158	200	35	88	82
214	134262	N	N	N	N	TINEA CRURIS	1.54	72	30.4	102	0.92	160	141	52	80	92
215	1054229	N	N	N	N	XP	1.8	86	26.5	86	0.89	197	158	33	148	88
216	1028445	N	N	N	N	INTERTRIGO	1.71	90	30.8	107	0.95	138	145	24	97	166
217	1054430	N	Y	N	N	INTERTRIGO	1.57	74	30	102	0.91	127	102	33	81	82
218	1043866	N	N	N	N	PYODERMA, ACROCHORDONS	1.6	73	28.5	96	0.87	203	298	36	135	105
219	1044710	N	N	N	N	ACROCHORDONS	1.57	72	29.2	104	0.92	157	116	47	96	92
220	1025863	N	Y	N	N	TINEA CRURIS ET CORPORIS	1.72	86	29.1	96	0.94	107	56	33	63	94

S No.	MRD	Name	Age (Years)	Gender	Chief Complaints												Duration (days)	Associated disease	Sites involved								
					itching	redness	swelling	maceration	pigmentation	striae	yellow raised	pus discharge	skin tags	scales	pain	others			Inguinal	Axilla	Face	Lower limb	Upper limb	scalp	Neck	Eye lids	Trunk
221	964286	S	54	F	N	N	N	N	N	N	Y	N	N	N	N	N	300d	N	N	N	N	N	N	N	N	Y	N
222	205361	GK	48	F	N	N	N	N	N	N	N	N	Y	N	N	N	60d	N	N	N	N	N	N	N	N	Y	N
223	61396	H	26	M	N	Y	Y	N	N	N	N	Y	N	N	N	N	5d	N	N	N	N	N	N	N	N	N	Y
224	1055836	PK	55	F	Y	Y	N	N	N	N	N	N	N	Y	Y	N	120d	N	N	N	N	N	N	N	N	N	N
225	738186	SK	39	F	N	N	N	N	N	N	N	N	Y	N	N	N	120d	N	N	N	N	N	N	N	N	Y	N
226	8022	MK	37	F	N	N	N	N	N	N	Y	N	N	N	N	N	30d	N	N	N	N	N	N	N	N	Y	N
227	260935	CL	64	M	N	Y	Y	N	N	N	N	Y	N	N	Y	N	3d	N	N	N	N	Y	N	N	N	N	N
228	991636	RG	61	M	Y	Y	N	N	N	N	N	N	N	Y	N	N	20d	DM II	N	N	N	N	N	N	N	N	N
229	5748	SP	50	F	N	N	N	N	N	N	N	N	Y	N	N	N	60d	N	N	N	N	N	N	N	Y	N	N
230	212595	RK	48	F	N	N	N	N	N	N	N	N	Y	N	N	N	90d	N	N	N	N	N	N	N	Y	N	N
231	216924	BS	55	M	N	N	N	N	N	N	N	N	Y	N	N	N	75d	N	N	N	N	N	N	N	Y	N	N
232	3333	S	43	F	N	Y	Y	N	N	N	N	Y	N	N	Y	N	4d	N	N	N	N	Y	N	N	N	N	N
233	1064862	SR	60	F	Y	Y	N	N	N	N	N	N	N	Y	N	N	12d	N	Y	N	N	N	N	N	N	N	N
234	5244	S	44	F	Y	Y	N	Y	N	N	N	N	N	N	Y	N	21d	DM II	Y	N	N	N	N	N	N	N	N
235	534878	M	48	F	Y	Y	N	N	N	N	N	N	N	Y	N	N	730d	N	N	N	N	N	N	N	N	N	N
236	82795	AA	46	F	Y	Y	N	Y	N	N	N	N	N	N	Y	N	21d	DM II	Y	N	N	N	N	N	N	N	N
237	1069921	PG	43	F	Y	Y	N	Y	N	N	N	N	N	N	Y	N	20d	DM II	N	N	N	N	N	N	N	N	N
238	1070602	G	60	M	N	N	N	N	N	N	N	N	Y	N	N	N	150d	N	N	N	N	N	N	N	Y	N	N
239	5868	GS	49	M	N	N	N	N	N	Y	N	N	Y	N	N	N	90d	N	N	Y	N	N	N	N	Y	N	Y
240	601973	BK	49	F	N	N	N	N	N	N	Y	N	N	N	N	N	60d	N	N	N	N	N	N	N	N	Y	N
241	1070750	RG	41	F	N	Y	N	Y	N	N	N	N	Y	N	Y	N	15d	DM II	Y	N	N	N	N	N	Y	N	N
242	256713	GS	48	M	Y	Y	N	Y	N	N	N	N	N	N	N	N	7d	DM II	N	N	N	N	N	N	N	N	N
243	1061921	CS	47	M	N	N	N	N	N	N	N	N	Y	N	N	N	60d	N	N	N	N	N	N	N	Y	N	N
244	1071132	AA	55	M	N	N	N	N	Y	N	N	N	N	N	N	N	30d	DM II	N	Y	N	N	N	N	Y	N	N
245	513585	PS	64	M	N	N	Y	N	N	N	N	Y	N	N	Y	N	5d	DM II	N	N	N	Y	N	N	N	N	N
246	1004541	MP	54	M	N	Y	N	Y	N	N	N	N	N	N	Y	N	10d	DM II	Y	N	N	N	N	N	N	N	N
247	934980	HK	57	F	N	N	N	N	N	N	Y	N	Y	N	N	N	45d	DM II	N	N	N	N	N	N	Y	Y	N
248	860865	JS	66	M	N	N	N	N	N	N	N	N	Y	N	N	N	60d	N	N	N	N	N	N	N	Y	N	N
249	811496	HS	78	M	N	N	N	N	Y	N	N	N	N	N	N	N	30d	N	N	N	N	N	N	N	Y	N	N
250	980217	SK	70	F	N	Y	N	Y	N	N	N	N	N	N	Y	N	14d	N	N	N	N	N	N	N	N	N	N

S No.	MRD	Inframammary	Buttocks	WEB SPACES	Any other	Diagnosis	Height(m)	weight(kg)	BMI(kg/m²)	waist circumference (cm)	waist-hip ratio	Fasting lipid profile				FBS(mg/dl)
												Total Cholesterol	TG	HDL	LDL	
221	964286	N	N	N	N	XP	1.58	76	30.4	104	0.92	215	179	49	131	102
222	205361	N	N	N	N	ACROCHORDONS	1.6	76	29.7	102	0.96	196	153	50	144	101
223	61396	N	N	N	N	FOLLICULITIS	1.8	86	26.5	91	0.94	167	186	47	105	84
224	1055836	N	N	N	PALM AND SOLES	PALMOPLANAR PSORIASIS	1.62	78	29.7	101	0.91	190	88	67	119	93
225	738186	N	N	N	N	ACROCHORDONS	1.6	74	28.9	98	0.96	186	46	60	109	101
226	8022	N	N	N	N	XP	1.65	73	26.8	96	0.94	172	116	51	117	86
227	260935	N	N	N	N	PYODERMA	1.75	78	25.5	96	0.96	118	105	35	76	78
228	991636	N	Y	N	N	TINEA CORPORIS	1.81	102	31.1	110	0.98	161	180	33	91	108
229	5748	N	N	N	N	ACROCHORDONS	1.6	75	29.3	102	0.94	361	250	61	269	95
230	212595	N	N	N	N	ACROCHORDONS	1.62	78	29.7	102	0.92	243	235	48	156	104
231	216924	N	N	N	N	ACROCHORDONS	1.76	88	28.4	112	1.05	163	184	29	75	83
232	3333	N	N	N	N	FOLLICULITIS	1.58	72	28.8	106	0.94	228	152	42	154	98
233	1064862	N	N	N	N	TINEA CRURIS	1.62	78	29.7	102	0.92	150	165	58	70	92
234	5244	N	N	N	N	INTERTRIGO	1.67	78	28	102	0.96	226	365	40	162	122
235	534878	N	N	N	PALM AND SOLES	PALMOPLANAR PSORIASIS	1.6	77	30.1	104	0.94	173	187	45	81	92
236	82795	N	N	N	N	INTERTRIGO	1.62	80	30.5	106	0.94	212	184	43	132	112
237	1069921	Y	N	N	N	INTERTRIGO	1.65	104	38.2	116	0.95	133	53	31	90	178
238	1070602	N	N	N	N	ACROCHORDONS	1.8	94	29	106	1.01	150	162	36	97	88
239	5868	N	N	N	N	ACROCHORDONS, STRIAE DISTENSAE	1.78	98	30.9	112	1.03	194	162	51	141	102
240	601973	N	N	N	N	XP	1.62	75	28.6	106	0.96	177	93	49	103	94
241	1070750	N	N	N	N	INTERTRIGO, ACROCHORDONS	1.62	114	43.4	116	0.95	170	136	37	118	118
242	256713	N	Y	N	N	INTERTRIGO	1.78	94	29.7	102	0.96	226	237	45	159	108
243	1061921	N	N	N	N	ACROCHORDONS	1.76	87	28.1	96	0.92	147	190	29	105	95
244	1071132	N	N	N	N	AN	1.75	98	32	112	1.05	219	147	35	167	126
245	513585	N	N	N	N	PYODERMA	1.8	88	27.2	102	0.96	176	70	71	98	117
246	1004541	N	N	N	N	INTERTRIGO	1.78	96	30.3	114	1.03	163	98	49	100	125
247	934980	N	N	N	N	ACROCHORDONS, XP	1.62	86	32.8	112	0.96	235	369	42	152	119
248	860865	N	N	N	N	ACROCHORDONS	1.74	82	27.1	102	0.98	103	185	38	49	87
249	811496	N	N	N	N	AN	1.77	93	29.7	98	0.92	115	50	42	61	88
250	980217	Y	N	N	N	INTERTRIGO	1.6	65	25.4	102	0.94	115	86	29	75	93

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Date 20/4/2021

The Principal,
DMC Ludhiana

Subject: Approval of Plan of Thesis.

This is to inform you that the Faculty of Medical Sciences in its meeting held on 22.01.2021 has approved the plan of thesis of **Amandeep Singh**, a student of your college for **MD Dermatology, Venerology & Leprosy** along with the names of Supervisor/Co-Supervisor as under:-

Topic Plan of Thesis
To study pattern of dermatological manifestations of obesity

Supervisor
Dr. Sunil Gupta
Prof. & Head,
Dept. of Dermatology,
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Co-Supervisor
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The student has to submit the three copies of thesis (along with PDF format) , to the Controller of Examinations, BFUHS, Faridkot, at least six months before the commencement of theory examination, if he/she is otherwise eligible to take the examination. He /She must directed to attach a photocopy of this letter with the forwarding letter while submitting thesis to the University along with the letter of approval of University for change of supervisor/Co-Supervisor, change of topic,(if any).

Dr. S P Singh MS(Oph)
Controller of Examinations

Note :

1. ***If any discrepancy found regarding topic of thesis or Supervisor/Co-Supervisor, inform undersigned within one month after issue of this letter.***
2. *The student may submit the thesis as per the schedule already conveyed i.e for May/June examination 30th Nov of the preceding the year of examination and for Nov/Dec examinations 31st May of the year of examination.*

TO STUDY PATTERN OF DERMATOLOGICAL MANIFESTATIONS OF OBESITY

THESIS PROPOSAL

FOR APPROVAL OF SUBJECT OF THESIS TO BE SUBMITTED

IN PARTIAL FULFILMENT OF THE REQUIREMENTS

FOR THE DEGREE OF

Doctor of Medicine

in

DERMATOLOGY, VENEREOLOGY AND LEPROLOGY

OF THE

BABA FARID UNIVERSITY OF HEALTH SCIENCES

FARIDKOT

November, 2019

Dr. Amandeep Singh

DEPARTMENT OF DERMATOLOGY, VENEREOLOGY & LEPROLOGY
DAYANAND MEDICAL COLLEGE & HOSPITAL, LUDHIANA
LUDHIANA, PUNJAB-141001

ABSTRACT OF PLAN OF THESIS

Title	To study pattern of Dermatological manifestations of obesity
For the degree of	M.D. Dermatology, Venereology & Leprology
Name of the candidate	Dr Amandeep Singh
Supervisor	Dr. Sunil Gupta, Prof. and Head, Department of Dermatology, DMCH, Ludhiana
Co-supervisor(s)	Dr. Sukhjot Kaur, Associate Professor, Department of Dermatology, DMCH, Ludhiana Dr. Naveen Mittal ,Professor, Department of Endocrinology, DMCH Ludhiana
Institution	Dayanand Medical College and Hospital, Ludhiana
University	Baba Farid University of Health Sciences, Faridkot

Introduction: Obesity is a major health problem associated with various skin manifestations. This study will be conducted to determine prevalence of various skin manifestations in obese population.

Aim of study: To study the pattern of dermatological manifestations in patients with obesity.

Materials and methods: The study will be conducted at Dayanand Medical College and Hospital, Ludhiana on patients with obesity. A total of 250 patients of obesity will be included in the study to find out the pattern of disease.

Clinical significance: To prevent long term cutaneous complications of obesity by early diagnosis and treatment.

Keywords: Obesity, Dermatological manifestations.

(Signature) Candidate	(Signature) Co-supervisor	(Signature) Co-supervisor	(Signature) Supervisor	(Signature) Principal

CERTIFICATE OF FACILITIES AVAILABLE

This is to certify that the facilities for work on the subject of thesis titled **"TO STUDY PATTERN OF DERMATOLOGICAL MANIFESTATIONS OF OBESITY"** exist in the Department of Dermatology, Venereology & Leprosy at Dayanand Medical College and Hospital, LUDHIANA and will be provided to the candidate. We will see that the data being included in the thesis are genuine and is collected by the candidate himself under our supervision and guidance.

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Place : Ludhiana

Date : 4/12/19

APPROVAL PROFORMA
BY
RESEARCH & ETHICAL COMMITTEE
DAYANAND MEDICAL COLLEGE & HOSPITAL, LUDHIANA

Name of candidate	Dr. Amandeep Singh
Department	DERMATOLOGY, VENEREOLOGY AND LEPROLOGY
Topic of Thesis	TO STUDY PATTERN OF DERMATOLOGICAL MANIFESTATIONS OF OBESITY
Likely date of appearing in PG examination	MAY-JUNE 2022
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Dr. Jaspal Singh (Professor & Head, Surgery)	Mr. A K Jindal (Member, Ethics Committee)
Dr. Dinesh Gupta (Professor & Head, Medicine)	Dr. Anu Sharma (Professor, Anatomy)
Dr. Sandeep Kaushal (Assistant Dean Academics)	Dr. Deepinder K Chhina (Professor & Head, Microbiology)
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Dr. Gurpreet S Wander (Vice Principal)	Dr. Gurpreet S Wander (Vice Principal)
	Dr. Gagandeep Singh (Secretary, Ethics Committee Professor & Head, Neurology)

Approved: Yes / ~~No~~

Dr. Sandeep Puri
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Approved: Yes / ~~No~~

Prof. Arvind Malhotra
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BABA FARID UNIVERSITY OF HEALTH SCIENCES, FARIDKOT

PROFORMA FOR REGISTRATION OF SUBJECT FOR DISSERTATION

1	NAME OF THE CANDIDATE AND ADDRESS	Dr. Amandeep Singh Guru Harkrishan Nagar ,Disposal Road, Opp. Sharma Dairy ,Khanna, Distt.- Ludhiana (Pb.)
2	NAME OF THE INSTITUTE	Dayanand Medical College & Hospital, Ludhiana.
3	COURSE OF STUDY AND SUBJECT	M.D. Dermatology, Venereology And Leprology
4	DATE OF ADMISSION OF COURSE	06-05-2019
5	TITLE OF THE TOPIC	TO STUDY PATTERN OF DERMATOLOGICAL MANIFESTATIONS OF OBESITY

6. BRIEF RESUME OF THE INTENDED WORK

6.1 NEED FOR THE STUDY

Obesity is a major 21st-century health problem that is predominantly considered a lifestyle disease associated with metabolic, genetic, social, behavioral, dietary, and cultural factors. The World Health Organization (WHO) classifies "overweight" as a body mass index (BMI) of 23-24.9 kg/m^2 and obesity as a BMI of≥25 kg/m^2 for Asian population. Morbidity and mortality in obese population is significantly more than the normal weight individuals.

Obesity increases the risk of coronary heart disease, hyperlipidaemia, osteoarthritis, diabetes and sleep apnoea. It is also directly linked with many other disorders like PCOD, Cushings syndrome,bulimia nervosa, GERD, fatty liver, cholelithiasis, colonic cancers, stroke, breast cancers and psychological problems like stress and depression.

Obesity causes abnormalities in several functions of the skin, including sebum production, skinbarrier function and sweat production. It also induces changes in lymphatics, collagen structure and function and subcutaneous fat.

Skin manifestations of obesity include acanthosis nigricans, acrochordon, keratosis pilaris, hirsutism, striae distensae, and redistribution of body fat. Acanthosis nigricans is the one of the common dermatological manifestation of obesity. Acrochordon is strongly associated with diabetes than with obesity.

Many infectious and inflammatory conditions like acne, folliculitis, intertrigo, hidradenitis suppurativa, psoriasis and cellulitis have been proved to be aggravated by co-existence of obesity.

Being aware of such clinical signs and the underlying systemic disorders may facilitate earlier diagnosis, thereby permitting early therapy initiation

and prevention of long-term sequelae of obesity. Understanding of these disorders is necessary both for the physicians as well as for the dermatologists, as many of these conditions are treatable, leading to an improved quality of life and reduction in psychological distress.

Thus, the present study is undertaken to shed light on the dermatological manifestations of obesity thereby helping in early treatment and prevention of long term complications of obesity, among the patients of different age groups attending the outpatient and inpatient Department of Dermatology and Endocrinology in Dayanand Medical College and Hospital, Ludhiana.

6.2 REVIEW OF LITERATURE

Obesity is now widely considered as a major public health problem. It is considered to be a global epidemic. Overweight and obesity are defined by WHO as abnormal or excessive fat accumulation that may impair health.[1]

In accordance with the consensus by WHO, as per "Report of WHO Consultation", normal BMI for Asians is 18.5-22 kg/m^2, overweight (BMI >23kg/m^2),at risk (BMI 23-24.9 kg/m^2), obesity I (BMI25-29.9 kg/m^2), obesity II (BMI >30 kg/m^2).[2]Obesity can also be assessed by using the waist circumference and waist-to-hip ratio.[3]

According to The National Institute of Diabetes, Digestive and Kidney Diseases (NIDDK), women with waist–hip ratios of more than 0.8, and men with more than 1.0, are at increased health risk because of their fat distribution. Cut off for waist circumference for South Asian men and women are more than 90 cm and more than 80 cm respectively. According to the Centres for Disease Control and Prevention-United States (CDC-US) growth charts, which are relatively specific, the 85th percentile is the cut off point for being overweight, whereas the 95th percentile is the cut off point for obesity.[4]

Over the past 30 years its prevalence has increased significantly worldwide.[5] In most of the Asian countries ,prevalence of obesity has increased many folds since last few decades.[5] Morbidity and mortality in obese population is significantly more than the normal weight individuals.[6] Waist circumference, waist–hip ratio and waist–height ratio, have been suggested as being superior to BMI in predicting CVD risk.[7]

4 million deaths worldwide are due to elevated BMI. Among the causes of mortality, cardiovascular events accounts for more than two third of the obesity related deaths. This necessitates the increasing need to identify

and screen people who have increased BMI for early initiation of treatment and reduction of mortality.[8]

Obesity carries a significant impact on psychological health, as well. It is indirectly labelled to anxiety, impaired social interaction and depression.[9] Obesity has a wide range of effects and contributes to the pathogenesis of metabolic, cardiovascular, carcinogenic, musculoskeletal and cutaneous disorders.[10]

Skin is one of the most important organ affected by obesity. On the molecular level, these changes are due to multiple interactions of hormonal factors, adipocytokines and mechanical factors. Most of the dermatological findings are correlated to the pathology of obesity. However, some are non-obesity related conditions. These skin conditions have a positive association with diabetes and insulin-resistance.[11,12]

Skin is affected in a number of different ways in obesity.[13] Apart from certain dermatoses that appear in obesity, certain cutaneous disorders are aggravated by obesity.[14]

The skin changes in obesity are due to increased weight leading to mechanical changes, hormonal imbalance like hyperandrogenism and insulin resistance leading to hyperinsulinemia. Altered physiological functions of skin secondary to obesity like changes in barrier function, excessive sweat and sebaceous glands secretion , an impaired lymphatic drainage, altered collagen structure and functioning, delayed wound healing , impaired micro and macrocirculation.[15-17] These changes lead to multiple cutaneous manifestations which are observed frequently in obese people.

Dermatological changes have been reported in patients with obesity, especially bacterial and fungal infections in skin folds due to occlusion, striae distensae due to skin stretching, stasis pigmentation related to peripheral vascular disease, acanthosis nigricans and skin tags secondary

to insulin resistance, and plantar hyperkeratosis due to increases weight.[12]

According to a study conducted in Lahore, common dermatoses seen in obese patients were acanthosis nigricans, striae, acrochordons, and various infections. Out of these acanthosis nigricans, acrochordons and striae had statistically significant relationship with obesity as compared to control group ($p<0.05$).[18]

A descriptive comparative study which was conducted in the Obesity Clinic of the Department of Physical Medicine and Rehabilitation in a tertiary care hospital of Kerala compared the skin manifestaions of hundred obese individuals above 12 years with that of hundred non obese individuals .The commonest skin manifestation was acanthosis nigricans. Features of hyperandrogenism and increased incidence of cutaneous infections were seen.[19]

In a study conducted at Karachi from 1st January 2014 till 30th June 2014, patients belonging to both sexes and different age groups having body mass index (BMI) >25kg/m^2 with cutaneous manifestations of obesity were enrolled. The most common finding observed was acanthosis nigricans (49%), followed by striae (17%), fungal infections (15%), acrochordons (12%), viral infections (11%), hirsutism (11%) and bacterial infections (7.5%). Other less common associations included: xanthomas, corns, plantar hyperkeratosis and acne.[20]

In a cross sectional descriptive observational study conducted at a tertiary hospital in Tamil Nadu, most individuals had mild obesity and elevated waist-hip ratio. Acanthosis nigricans was the most common cutaneous manifestation present among 95% individuals. The other common cutaneous manifestations were striae distensae, acrochordons, intertrigo, dermatophytosis and folliculitis.[21]

A study conducted in Egypt showed presence of skin tags in 64.2% of obese patients as compared to 52.1% in nonobese patients.[22]

A study by Garcia et al showed increased appearance of skin tags with increase in severity of obesity.[23]

The 'PROCEED' study by Wolf et al showed that obese individuals had 8% increase in soft tissue infections compared to non obese.[24]

6.3 AIMS AND OBJECTIVES OF THE STUDY

1. To study the pattern of various dermatological manifestations in patients with obesity.

7. MATERIALS AND METHODS

7.1 Source of Data

Patients attending outpatient and inpatient in Department of Dermatology and Endocrinology of Dayanand Medical College and Hospital, Ludhiana will constitute the study material and the source of data.

7.2 METHOD OF COLLECTION OF DATA

INCLUSION CRITERIA:

Patients aged >18 years,belonging to either sexes attending outpatient and inpatient in Department of Dermatology and Endocrinology of Dayanand Medical College and Hospital, Ludhiana, will be included in the study.

EXCLUSION CRITERIA:

- Patients not willing to participate
- Pregnant women and patients on systemic corticosteroids will be excluded from the study.

METHODS:

A total of two hundred and fifty clinically diagnosed cases of obesity will be included in this study.After informed consent, a detailed history, a comprehensive general and systemic examination will be carried out followed by a detailed skin examination (including hair, scalp and nails). The demographic details of the enrolled patients will be documented. Height (meters) and weight (kg) will be measured. BMI to be calculated using the formula weight (kg) / height$(m)^2$. Patients having BMI >25 will be included in the study. Cases will be labelled as obesity grade I and obesity grade II as per classification of BMI by WHO for Asians. Cutaneous lesions will be diagnosed clinically.

A detailed physical examination including recording of vitals (pulse rate, blood pressure); anthropometry measurements-height (in meters), weight (in kgs), waist circumference in cms (at level of umblicus) and hip circumference in cms (at the level of widest part of buttocks) will be done.

Relevant laboratory investigations like complete blood count, fasting lipid profile, random blood sugar and Special tests like Wood's lamp examination, scraping for fungus will be performed when required.

Number of cases

Two Hundred and fifty

Statistical Analysis

Data will be described in terms of range; mean ±standard deviation (± SD), frequencies (number of cases) and relative frequencies (percentages) as appropriate. All statistical calculations will be done using SPSS (Statistical Package for the Social Science) SPSS 21 version statistical program for Microsoft Windows.

7.3 DOES THE STUDY REQUIRE ANY INVESTIGATIONS OR INTERVENTIONS TO BE CONDUCTED ON PATIENTS OR OTHER HUMANS OR ANIMALS? IF SO, PLEASE DESCRIBE BRIEFLY.

The following investigations will be advised to patients.

1. Fasting lipid profile

2. Fasting blood sugar

The study does not require any animal experimentation and/or human intervention.

7.4 HAS ETHICAL CLEARANCE BEEN OBTAINED FROM YOUR INSTITUTION IN CASE OF 7.3?

Attached herewith.

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PROFORMA

S.NO.-

Name : Date:

MRD No.:

Age: Sex:

Father/Husband's name:

Occupation:

Marital Status:

Address:

Contact No:

Diagnosis:

Chief complaint:

History of any Associated illness:

Signature of Candidate **Signature of Supervisor**

General Physical examination:

Height(m) Weight(kg) BMI(kg/m^2)

Systemic Examination:

Cutaneous manifestation:

- Acanthosis nigricans
- Striae
- Fungal infections
- Acrochordons
- Viral infections
- Hirsutism
- Bacterial infections
- Xanthomas
- Intertrigo
- Any Other

Morphology of Lesions:

Sites of Lesion:

Distribution:

Investigations :

FBS

Fasting lipid profile

Signature of Candidate **Signature of Supervisor**

INFORMED PATIENT CONSENT

Date: _____

I.....D/o, W/o, S/o.....

.....am willing to participate in the study **"TO STUDY PATTERN OF DERMATOLOGICAL MANIFESTATIONS OF OBESITY"** I have fully understood that no special tests and extra expenditure in any form, merely for the purpose of thesis is being carried out. I will not be paid to participate in study. I am free to opt out the study any time without assigning any reason.

.....
Signature of the Witness

Name:

.....
Signature of the patient

Signature of Supervisor

Signature of Candidate

मरीज अनुमति फार्म

तिथि _____

मैं _____ पत्नी/पुत्र/पुत्री _____

निवासी _____ इस शोधकार्य ""**TO STUDY PATTERN OF DERMATOLOGICAL MANIFESTATIONS OF OBESITY**" में भाग लेने के लिए सहमत हूँ। मैं इस शोधकार्य में भाग लेने के लिए अपनी स्वेच्छा से तैयार हूँ। मैं पुष्टि करता/करती हूँ की यह स्वीकृति देने के लिए मुझे कोई आर्थिक प्रलोभन नहीं दिया गया और न ही दबाव डाला गया है। मुझे इस शोधकार्य का उद्देश्य स्पष्ट कर दिया गया है और मुझे यह विश्वास है की यह विज्ञान और मानवता के लाभ के लिए है।

मैं इस शोधकार्य में भाग लेने के सम्बन्ध में किसी भी समय अपनी अस्वीकृति व्यक्त कर सकता/सकती हूँ और मेरे अस्वीकृति व्यक्त करने का मेरे इलाज पर कोई प्रभाव नहीं पड़ेगा।

_____ _____

गवाह के हस्ताक्षर रोगी के हस्ताक्षर/अंगूठे का निशान

नाम: नाम:

_____

अन्वेषक के हस्ताक्षर

_____

विद्यार्थी के हस्ताक्षर

ਮਰੀਜ਼ ਰਜ਼ਾਮੰਦੀ ਫਾਰਮ

ਮਿਤੀ_____

ਮੈ_____ਪਤਨੀ/ਪੁੱਤਰ/ਪੁੱਤਰੀ_____

ਵਾਸੀ _____

"TO STUDY PATTERN OF DERMATOLOGICAL MANIFESTATIONS OF OBESITY" ਤੇ ਚਲ ਰਹੀ ਖੋਜ ਵਿਚ ਹਿੱਸਾ ਲੈਣ ਲਈ ਰਜ਼ਾਮੰਦ ਹਾਂ । ਇਸ ਖੋਜ ਕੰਮ ਵਿਚ ਹਿੱਸਾ ਲੈਣ ਲਈ ਮੈ ਆਪਣੀ ਮਰਜ਼ੀ ਨਾਲ ਤਿਆਰ ਹਾਂ । ਮੈ ਇਹ ਦਸਣਾ ਚਾਹੁੰਦਾ ਹਾਂ/ਚਾਹੁੰਦੀ ਹਾਂ ਕੇ ਇਹ ਰਜ਼ਾਮੰਦੀ ਦੇਣ ਵਾਸਤੇ ਮੈਨੂ ਕਿਸੇ ਵੀ ਤਰਾਂ ਦਾ ਲੋਭ-ਲਾਲਚ ਨਹੀ ਦਿੱਤਾ ਗਿਆ ਅਤੇ ਨਾ ਹੀ ਕੋਈ ਦਬਾ ਪਾਇਆ ਗਿਆ ਹੈ ।ਮੈਨੂ ਇਸ ਖੋਜ ਦਾ ਮਨੋਰਥ ਸਮਝਾ ਦਿੱਤਾ ਗਿਆ ਹੈ ਅਤੇ ਮੈਨੂ ਇਹ ਭਰੋਸਾ ਦਵਾਇਆ ਗਿਆ ਹੈ ਕੇ ਇਹ ਖੋਜ ਵਿਗਿਆਨ ਅਤੇ ਮਾਨਵਤਾ ਦੀ ਭਲਾਈ ਲਈ ਹੈ ।

ਮੈ ਇਸ ਖੋਜ ਕੰਮ ਵਿਚ ਹਿੱਸਾ ਲੈਣ ਬਾਰੇ ਆਪਣੀ ਰਜ਼ਾਮੰਦੀ ਕਿਸੇ ਵੀ ਸਮੇ ਵਾਪਸ ਲੈ ਸਕਦਾ ਹਾਂ/ ਸਕਦੀ ਹਾਂ ਅਤੇ ਮੇਰੀ ਰਜ਼ਾਮੰਦੀ ਵਾਪਸ ਲੈਣ ਦਾ ਮੇਰੇ ਇਲਾਜ ਤੇ ਕੋਈ ਅਸਰ ਨਹੀ ਹੋਵੇਗਾ।

_____ ਗਵਾਹ ਦੇ ਹਸਤਾਖਰ

_____ ਮਰੀਜ਼ ਦੇ ਹਸਤਾਖਰ

ਮਿਤੀ_____

ਮਿਤੀ_____

ਪਤਾ _____

ਪਤਾ _____

_____ ਨਿਰੀਖਕ ਦੇ ਹਸਤਾਖਰ

_____ ਸਿਖਿਆਰਥੀ ਦੇ ਹਸਤਾਖਰ